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TUBERCULOSIS IN INFANCY*

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It is not my purpose in this paper to do more than discuss certain limited aspects of the subject of tuberculosis in the first two years of life. The literature of tuberculosis is so enormous, and appears so complete, that it is difficult to add anything really new to our knowledge of this subject. In looking over the literature I have been impressed with the fact that in many articles the conclusions drawn are not supported by the final proof afforded by postmortem examination. This is true especially in this country, where the difficulty of obtaining permission to make postmortems on infants is so great, that it has been difficult to record observations on even a small series of cases in which final proof was obtained. The new Memorial Building of the Infants' Hospital has been open for nineteen months. During the last nine months of this time the giving of written permission for autopsy in case of death has been a necessary requirement when babies are admitted to the hospital. The increase in the number of our postmortems under this rule has been the impetus which caused me to reopen the much discussed subject of tuberculosis.

The importance of tuberculosis as a cause of death in the first two years of life is shown by the fact that in a series of sixty-two successive necropsies, tuberculosis was found in twenty-five, and was considered the direct cause in twenty-three cases, and as the contributing cause in the other two.

THE SOURCE AND MODE OF INFECTION

Of all the subjects of discussion in connection with tuberculosis in early life, the one which his occupied most space in the literature is the question of the source of the infection, and its portal of entry into the body. This question is still one about which extremely divergent views are held. One set of writers believes that milk infected with the bovine type of bacillus is the most common cause of the disease in infancy, and

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* Read before the joint meeting of New York, Philadelphia and New England Pediatric Societies, Philadelphia, Nov. 9, 1915.

that the most common portal of entry is the intestinal tract, or the tonsils. Another group of writers believes that the human type of bacillus is the most common cause, and that the portal of entry is usually the lungs.

The evidence brought forward in support of these two views is of varying character. Such evidence as is based on statistics of the frequency of feeding with unsterilized milk, or of the frequency of known exposure to tuberculosis is only of confirmatory value. The two most important forms of evidence bearing on this question are, first, that derived from bacteriologic study of the bacilli obtained from infants by culture or animal inoculation, and second, that derived from a careful study of the lesions found postmortem.

Based on this evidence, the reports of various investigators remain extremely contradictory. So eminent an authority as von Pirquet[1] states in his article on tuberculosis in Feer's textbook, that the human bacillus, entering through the lungs, is the cause of tuberculosis in infancy, basing his conclusions on the 1,060 careful autopsies by Albrecht, in which a primary intestinal lesion was found in seven cases only, and on the 189 necropsies of Ghon, in which all but three showed what Ghon considered a primary lesion in the lung. On the other hand Mitchell's[2] studies in Edinburgh showed that in 90 per cent. of the cases the bacillus was one of the bovine type.

In general the reports of postmortem findings show a greater frequency of involvment of the peribronchial and mediastinal lymph nodes than of the mesenteric. Some prominent advocates of milk infection have endeavored to explain this on the theory that the bovine bacillus can pass without causing a lesion, not only through the intestinal mucous membrane, but even through the mesenteric lymph nodes, locating itself in the lymph nodes of the chest as the point of minimum resistance. This theory I believe to be inadequately supported, and entirely untenable. It may be possible that the tubercle bacillus can enter the body of the infant without a lesion at the portal of entry; this is still an open question. But that the bacillus can skip one set of lymph nodes to locate in another, is highly improbable.

The contradictory character of the evidence as to the source of infection and mode of invasion in infantile tuberculosis can only be explained on the ground of a variation according to locality. Such variation is probably largely dependent on the character of milk supplies, and the frequency of tuberculous disease in cattle in various parts of the world. I believe that infants can be infected either with human

1. Feer: Lehrbuch der Kinderheilkunde, 1914.
2. Mitchell: Brit. Med. Jour., 1914, No. 2768, p. 125.

bacilli, usually entering through the lungs, or with bovine bacilli, usually entering through the intestine. The present study of postmortem material therefore bears only on conditions in Boston and its vicinity.

The Primary Lesion.—Von Pirquet, in his recent textbook article, adheres to the view of Ghon, that tuberculous infection always shows a primary lesion at the portal of entry. This view is very different from the one which has been most generally accepted, which holds that the tubercle bacillus in infancy can, and usually does, pass through the tissues at the portal of entry, and then, following the lymph channels, reaches the lymph nodes, where it produces the primary lesion. This widely taught view is based on the rarity in infancy of phthisis, the chronic process most characteristic of adults, on the prominence of lymph node tuberculosis as the chronic process most characteristic of early life, and on the fact that the most prominent tuberculous lesions found in the lungs of infants are of a secondary nature. I have been inclined toward this view of tuberculosis in infancy, and have been teaching it to my students year after year.

In the recent series of necropsies at the Infant's Hospital, I determined to investigate this question of the primary lesion, by making most careful and thorough examination of the lungs and intestine in each case. It seemed possible that the primary lesion in the lung as described by Ghon and Albrecht might have been overlooked. Frequently at postmortems the lungs are examined no more carefully than by making one or two incisions in each lobe, which disclose the secondary lesions, miliary tuberculosis, or tuberculous bronchopneumonia, so prominent in infancy. It is then easy to assume that the caseous bronchial lymph nodes represent the primary lesion. In my series the lungs were cut up into the smallest fragments in the search for the primary lesion, and all suspected areas were subjected to microscopic examination.

The question of interest in this investigation was how often there would be found a lesion at the portal of entry recognizable as the primary lesion described by Ghon and Albrecht, and differing from the well-known secondary lesions.

As a result a primary lesion was actually found at the portal of entry in twenty-two of the twenty-five postmortems. The three cases in which a primary lesion was not found at the portal of entry all showed chronic tuberculosis of the bronchial lymph nodes, but none of the mesenteric lymph nodes. The nature of the primary lesion found in the lung in some cases was such as to suggest that it might easily have been overlooked, and that in the three cases a still more inconspicuous primary lesion may, in fact, have escaped observation.

Wollstein and Bartlett,[3] in a study of the tuberculous lesions found postmortem in 178 infants and young children, found tuberculous areas in the bronchial lymph nodes without pulmonary involvement in seven cases, and believe that this shows that it is possible for the tubercle bacillus to pass through the lungs without localizing there. They prefer this explanation to that of Ghon, who believes that in these cases the pulmonary lesion is overlooked. As a result of my observations I am inclined to revise my views on tuberculosis in infancy, to differ with Wollstein and Bartlett's explanation, and to believe with Ghon, that it is more probable that when a primary lesion is not found at the portal of entry it is overlooked.

The Location of the Primary Lesion.—The location of the primary lesion is important as evidence of the most frequent source of tuberculous infection in infants in Boston and its vicinity. The primary lesion was found in the lung in twenty cases, and in the intestine in two cases. If we add to the twenty cases the three in which the only chronic lesions were found in the bronchial lymph nodes we have a ratio of twenty-three cases of infection through the lung to two cases of infection through the intestine. This suggests that in Boston, at least, the most frequent mode of infection is by the inhalation of tubercule bacilli from a human source, but that infection through the ingestion of bovine bacilli in contaminated milk may occur.

The Primary Lesion in the Lung.—The lesion found in the lung seemed quite easily recognizable as a primary lesion, and distinguishable from the common widespread secondary lesions. In most cases it was a circular area, varying in size from that of a pea to that of a hazlenut. The center was caseous, but usually of a grayer color than the yellowish-white caseation seen in the secondary lesions. In some there was caseous softening, or small cavities; in one there was calcification. In a few cases the primary lesion was much larger, reaching to the pleura, where its situation was marked by firm fibrous adhesions. In two of these larger lesions there were large cavities, with irregular grayish walls. Around the periphery of some of the smaller lesions were numerous miliary tubercles, suggesting direct extension. In only one case was there more than one lesion of this description.

Microscopically the primary lesion differed from caseous tubercles and secondary caseous areas in this particular, that in the latter, even when the section showed wholly necrotic tissue, the lung structure, the outlines of bronchioles and alveoli, were faintly discernible. In the primary lesion the necrotic tissue always appeared uniform and the alveolar outlines could not be made out. In only one case was there evi-

3. Wollstein, M., and Bartlett, F.: Am. Jour. Dis. Child., 1914, viii, 362.

dence of healing as shown by fibrous tissue formation in the primary lesions, but this is to be explained by the fact that all the cases were fatal ones in young infants in whom the resistance was probably very low.

The situation of the primary lesion in the lung was as follows:

Left upper lobe	5 cases
Left lower lobe	0 cases
Right upper lobe	8 cases
Right lower lobe	5 cases
Right middle lobe	2 cases

The favorite situations were at the apex and near the hilus.

The Primary Lesion in the Intestine.—The lesion found in the intestine in two cases, and considered primary, was a tuberculous ulceration. In one case there was a single ulcer, while in the other case the ulcers were multiple. There was nothing about the appearance of these ulcerations differing from that seen in the secondary ulcerations found in several cases. My reasons for believing the intestinal ulceration to be a primary lesion in these two cases are, first, because in them the mesenteric lymph nodes *only* were tuberculous, and they were the only cases in which tuberculosis of the mesenteric lymph nodes was found without tuberculosis of the bronchial lymph nodes. As additional evidence, one of these cases was one of a series known to have occurred in a limited locality, and all having the same milk supply. Furthermore, animal inoculation from this case gave a bovine type of bacillus.

Involvment of the Lymph Nodes.—The bronchial lymph nodes were found tuberculous in the twenty cases having a primary lesion in the lung, and in three cases in which no primary lesion was found. They were involved alone in sixteen cases, including the three without evident pulmonary lesion. The mesenteric lymph nodes were tuberculous in addition to the bronchial lymph nodes in seven cases having a primary lesion in the lung, and were involved alone in the two cases having a primary lesion in the intestine.

Secondary Ulcers of the Intestine.—Tuberculous ulceration of the intestinal mucous membrane was found in seven cases beside the two in which this lesion was considered primary. In all cases the ulcers were multiple. My reasons for believing these lesions to be secondary are the following: 1. In all the seven cases a primary lesion was found in the lung. 2. In all of them there was tuberculosis of the bronchial lymph nodes, which in most cases appeared to represent a more advanced and older process than that in the mesenteric lymph nodes. 3. In all of them there was evident in the primary pulmonary lesion either caseous softening, or cavity formation, and conversely, in

no case in which such a condition was found in the primary lesion did I fail to find ulceration in the intestine. I believe it to be a probable conclusion that these tuberculous ulcerations of the intestine are secondary to swallowed tuberculous material coughed up from the softening of the primary lesion.

These seven cases with secondary tuberculous ulceration of the intestine were identical with the seven cases in which the mesenteric lymph nodes were involved in addition to the bronchial lymph nodes. This is a finding of the greatest importance in connection with the question of the ability of the tubercle bacillus to pass through the mucous membrane without causing a lesion at the portal of entry. In all there were nine cases in which the mesenteric lymph nodes were found to be tuberculous, and in all of them there was also found tuberculous ulceration of the mucous membrane of the intestine. There was no case in my series showing tuberculosis of the mesenteric lymph nodes without an intestinal lesion. In two cases the intestinal lesion was primary; in the other seven secondary to a primary lesion in the lung, from which tuberculous material was swallowed. I believe these findings to be evidence against the view of the ability of the tubercle bacillus to pass through the intestinal mucosa without leaving traces of its progress in the form of a definite lesion.

Other Secondary Lesions.—Chronic tuberculosis of the lymph nodes was found in every case. Most of the other secondary lesions were of an acute character. A more or less generalized miliary tuberculosis, or tuberculous meningitis was found in twenty-three cases. In two cases were chronic lesions only found. Tuberculous peritonitis was found in two cases. Solitary tubercle of the brain was found in one case.

The secondary lesions found in the lung were of two distinct kinds. One was a tuberculous bronchopneumonia characterized by exudate into the pulmonary alveoli, with more or less necrosis or caseation. This lesion was found in ten cases, and I believe it to represent an extension of the tuberculous process from the primary lesion to the lung through the bronchial channels. The other secondary lesion found in the lungs was a miliary tuberculosis, which both in macroscopic and microscopic appearance differed entirely from the tuberculous bronchopneumonia just described. The lesion consisted of numerous sharply defined miliary tubercles without any exudate into the surrounding alveoli. This was the sole secondary lesion found in the lung in thirteen cases. But such miliary tubercles were also found in a certain number of cases in which tuberculous bronchopneumonia was the most prominent lesion. This lesion probably represents extension of the tuberculous process from either the primary lesion or some sec-

ondary lesion through the medium of the general circulation, forming part of a general miliary tuberculosis. Twenty-three of the twenty-five cases showed miliary tubercles in various parts of the body.

The brain was not examined in three cases, and no lesion of the meninges was found in two cases. In nineteen cases a tuberculous meningitis was found, which was the immediate cause of death. In thirteen of these cases the meninges showed small miliary tubercles without evidence of exudate. In six cases, in addition to the miliary tubercles there was more or less cellular or fibrinous exudate along the course of the vessels.

Bacteriological Findings.—In only five cases in this series were animal inoculations made to determine whether the infection was with a human or with a bovine type of bacillus. The bovine type was found in one case, and the human type in four cases. The case in which the bovine type of bacillus was found was one of those having a primary lesion in the intestine and the one known to have been fed with contaminated milk. The four cases in which a human type of bacillus was found included one of the cases in which there was no apparent primary lesion. The cases in which the type of bacillus was tested by animal inoculation are too few to afford more than confirmatory evidence of the greater frequency of infection from a human source.

Previous Feeding.—The previous feeding of these patients was as follows: Fourteen babies had had milk of an unknown or doubtful character. Five babies had been fed exclusively on a milk considered to be of the best character. Six babies had been exclusively breast fed.

Exposure to Tuberculosis.—In four cases only was there a definite history of exposure to tuberculosis obtained. In a few others there was suspicion of such exposure.

THE DIAGNOSIS OF TUBERCULOSIS IN INFANCY

In this series of cases a diagnosis of the tuberculous nature of the disease was made during life in twenty-three out of twenty-five cases. This does not mean that the diagnosis of tuberculous infection in infancy is easy, or that it can be made in so large a number of cases. It must be remembered that these were all fatal cases, seen in the last stages of the disease. In fifteen of them a diagnosis was possible because they showed the definite clinical picture of tuberculous meningitis with characteristic cerebrospinal fluid. The two cases in which the diagnosis was not made were apparently cases of chronic nutritional disturbance with atrophy. One of them is particularly interesting. The child was treated in the hospital for some weeks as a feeding case and did very well, gaining weight steadily. At no time during its stay in hospital did it show any symptoms suggesting tuberculosis, nor did

it have any fever. Within twenty-four hours of its discharge from the hospital it had a series of convulsions followed by death. It was brought back for necropsy and showed extensive chronic tuberculosis of the bronchial lymph nodes with acute miliary tuberculosis of all the organs of the body, but none of the meninges. I am inclined to doubt if acute miliary tuberculosis can often be diagnosed in infancy unless the brain is involved.

The principal diagnostic feature in this series of cases was due to the existence of a tuberculous meningitis. The important question is, whether tuberculosis could have been recognized before the onset of this acute manifestation? For this is an important point in the treatment of the disease.

There are three important points beside the existence of such definite manifestations as tuberculous meningitis or tuberculous peritonitis, on which the diagnosis of tuberculosis in infancy is based. These are: 1. The finding of physical signs suggesting tuberculosis in the chest. 2. The evidence given by the tuberculin reaction. 3. The evidence by Roentgen-ray examination of the chest. An effort was made at the Infants' Hospital to carry out as thoroughly as possible these three methods of investigation, but owing to the fact that many babies were brought in in a moribund condition and died so soon after admission, the records have been very incomplete.

Physical Signs in the Lungs.—The physical examination of the lungs for signs suggesting tuberculosis gave the following results:

Positive	14 cases
Negative	11 cases

In four of the fourteen positive cases d'Espine's sign was the only one found. In the other ten cases evidences of consolidation in various parts of the lung were found which were of such a character as to suggest the possibility of tuberculosis, even if tuberculous meningitis had not also been present. In comparing the physical signs with the postmortem findings the following points were striking: 1. Acute miliary tuberculosis of the lung without tuberculous bronchopneumonia appears to give no recognizable physical signs in the lung. 2. Tuberculous bronchopneumonia usually does give recognizable physical signs in the lungs. In some cases only scattered râles are found; in others there are signs of consolidation in various parts of the chest. The infants may or may not show the appearance of bronchopneumonia with rapid breathing, etc. 3. The presence of d'Espine's sign in infancy usually means tuberculosis of the bronchial lymph nodes, but this sign may not be obtained in cases in which the glands are found enlarged at necropsy. 4. The primary lesion gives no recognizable physical signs in the chest,

unless very large, and in such a case the signs are only those of consolidation and are indistinguishable from tuberculous bronchopneumonia.

The Tuberculin Reaction.—Only the von Pirquet cutaneous reaction was employed. The results were as follows:

Positive	6 cases
Negative	12 cases
Not done	7 cases

The large number of negative reactions in cases proved to be tuberculosis is striking. It must be remembered, however, that many of the patients in the series were acutely ill, and that the reaction was observed very shortly before death, when resistance to the infection was probably at a minimum. It is possible that the results obtained in this series of cases has little bearing on the value of the reaction in cases not in the acute stage of the disease. Nevertheless, one of the chronic cases showed a persistently negative reaction, while in some of the most acute cases a positive reaction was observed within a very short time before death. On the whole, I am inclined to believe that the value of the cutaneous tuberculin reaction in infancy is not so great as many writers and statistical reports would lead us to suppose. A negative reaction seems to be a frequent finding in any stage of the disease, and gives no evidence at all. A positive reaction on the other hand, in infancy, means tuberculosis. For some time at the Infants' Hospital a routine von Pirquet test has been made on every case. We have yet to see a case in which a positive reaction has accompanied a negative postmortem finding. There was no case in this series in which a positive von Pirquet would have been the only means of diagnosis, if found at a time preceding the acute stage of the disease. There was one case in which the diagnosis rested on a positive von Pirquet with a positive d'Espine's sign.

Roentgen Ray Examination of the Chest.—This was made in thirteen cases of the series, and was positive in every case. There were several cases in which it gave the only positive finding. In general there was a surprisingly close correspondence between the Roentgen-ray picture and the postmortem findings. The enlargement of the bronchial lymph nodes could be made out in most of the cases. Tuberculous bronchopneumonia was correctly diagnosed from the Roentgen-ray in several cases, but not in all those in which it was found postmortem. The primary lesion was not shown, when small, but was conspicuous when very large. Miliary tuberculosis of the lung without tuberculous bronchopneumonia was not shown.

There were two cases in the series in which before the acute stage the diagnosis could have been made from the Roentgen-ray picture

alone, and two cases in which d'Espine's sign was positive, but in which the diagnosis of the pulmonary condition rested chiefly on the roentgenogram.

The table shows the occurrence of the three forms of diagnostic evidence in the series:

CERTAIN DIAGNOSTIC SIGNS IN TWENTY-FIVE CASES OF TUBERCULOSIS IN INFANCY, COMING TO NECROPSY

Clinical Type	Roentgen Ray	Signs in Lung	Von Pirquet
1. Tb. peritonitis	?	0	?
2. Atrophy	?	0	?
3. Tb. meningitis	?	0	?
4. Tb. meningitis	?	0	?
5. Malnutrtion	+	d'Espine only	0
6. Bronchopneumonia	+	+	0
7. Tb. meningitis	?	0	?
8. Tb. meningitis	?	0	0
9. Tb. meningitis	+	+	+
10. Tb. meningitis	?	+	+
11. Tb. meningitis	+	0	0
12. Tb. meningitis	+	0	0
13. Tb. meningitis	+	d'Espine only	?
14. Chronic tuberculosis of lung	+	+	0
15. Tb. meningitis	?	d'Espine only	+
16. Tb. meningitis	+	+	+
17. Tb. meningitis	?	0	0
18. Bronchopneumonia	+	+	0
19. Bronchopneumonia	?	0	?
20. Tb. meningitis	+	+	+
21. Chronic tuberculosis of lung	+	+	+
22. Tb. meningitis	+	+	0
23. Atrophy	?	0	0
24. Tb. peritonitis	+	+	0
25. Tb. meningitis	?	+	0

TYPES OF HYDROCEPHALUS—THEIR DIFFERENTIATION AND TREATMENT*

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The physiology of the cerebrospinal fluid, its origin, secretion and absorption, has been the object of intensive study during recent years.[1] The subject has been studied from many angles and by many observers, and the information thus afforded has opened a new vista into the diseases of the central nervous system, in which the cerebrospinal fluid plays an important part. It has been proved conclusively that the cerebrospinal fluid is, for the most part, at least, a secretory product of the choroid plexus. We have found, moreover, that the secretory activity of this gland may be influenced by certain factors, and in the course of our investigations it was found that thyroid extract had a distinctly *inhibitory* effect on the output of cerebrospinal fluid—a fact which may be of real practical importance in the treatment of conditions in which excessive secretion is the disturbing element. Many theories have been advanced as to the exact mode of absorption, but complete unanimity of opinion has not as yet been reached. Certain it is, however, that all abnormal accumulations of fluid within the ventricles are due to a loss of balance between secretion and absorption, the underlying factors being an obstruction to the outflow of the fluid from the ventricles, delayed or defective absorption, or a hypersecretion.

Of all the conditions in which the excessive accumulation of fluid has to be reckoned with, e. g., meningitis, brain tumor, and hydrocephalus, the latter alone, will be considered in this paper. The time has come to throw aside the crude classification of all forms of hydrocephalus, into internal and external: internal hydrocephalus clearly

* Submitted for publication Nov. 4, 1915.

1. The reader is referred to the contributions of Dixon and Halliburton, Thomas, Dandy and Blackfan, Weed and Cushing, and of Frazier and Peet as follows:

Frazier, C. H., and Peet, M. M.: Factors of Influence in the Origin and Circulation of the Cerebrospinal Fluid, Am. Jour. Physiol., 1914, xxxv, 3; The Action of Glandular Extracts on the Secretion of Cerebrospinal Fluid, Ibid., 1915, xxxvi, 4; Influence of Diiodotyrosine and Iodothyrine on the Secretion of Cerebrospinal Fluid, Ibid., 1915, xxxviii, 1.

Frazier, C. H.: The Cerebrospinal Fluid in Health and Disease, Jour. Am. Med. Assn., 1915, lxiv, 1119.

enough an obstructive form, and external hydrocephalus everything and anything not included in the "internal" variety. From our investigations, therefore, we venture to suggest a new classification, having a physiologic background with direct clinical application:

I. Hydrocephalus obstructivus.
II. Hydrocephalus nonabsorptus.
III. Hydrocephalus hypersecretivus.
IV. Hydrocephalus occultus.

I. In *Hydrocephalus obstructivus,* the internal hydrocephalus of the old nomenclature, there is mechanical obstruction to the natural drainage of the cerebrospinal fluid from one or more ventricles into the subarachnoid space, where the absorption takes place. This obstruction may be due to a congenital defect, such as absence of the aqueduct of Sylvius, or as is more frequently the case, it may be the result of adhesions from a preexisting inflammatory lesion. If the aqueduct of Sylvius is lacking or closed by adhesions, there will be a dilatation of both the third and the lateral ventricles, while a closure of the forearm of Munro would cause merely an enlargement of the lateral ventricle on the affected side. If, as is often true in cases of high-grade, but evenly-distributed hydrocephalus, the passage of the fluid through the foramina of Magendie and Luschka is blocked, there will be a general dilatation in which all the ventricles participate. While we now know that obstruction is not responsible for as large a number of cases of hydrocephalus as was formerly supposed, nevertheless it has been proved conclusively both by necropsy findings, and by the experimental production of hydrocephalus through occlusion of the aqueduct of Sylvius, that obstruction is one of the causative factors. Too great emphasis, however, can scarcely be laid on the importance of differentiating *Hydrocephalus obstructivus* from the other types in which the passageways from the ventricles are patulous.

II. In *Hydrocephalus nonabsorptus,* absorption is delayed or defective as has been proved by the phenolsulphonephthalein test. Whether the restricted absorption is to be attributed to (1) the cutting off of part of the subarachnoid space by adhesions, (2) a toxic substance in the fluid which prevents its absorption by the venous channels, or (3) whether it is due to an abnormal condition of the agents—whatever we grant them to be—which transport the fluid to the venous circulation, is still a matter of conjecture. Since the cerebrospinal fluid is absorbed by the venous channels, it is quite possible that an obstruction to the venous circulation, such as thrombosis, might be responsible for the delayed absorption. Indeed, Dandy and Blackfan succeeded in producing a low-grade experimental hydrocephalus by ligation of the vena galena magna. But we cannot entirely eliminate

the possibility of a change in the character of the fluid itself or an abnormal condition of the conveyors of the fluid to the venous circulation until there have been more pathologic examinations, and more chemical, bacteriologic and cytologic examinations of the fluid in this type. However this may be, *Hydrocephalus nonabsorptus* should be carefully differentiated from the other forms that an effective means may be instituted for draining the fluid to other areas where it may be absorbed.

III. By a process of elimination and by a careful consideration of the normal physiology of the cerebrospinal fluid and of the possible changes under abnormal conditions, the third type with apparent excessive accumulation of fluid has been attributed to hypersecretion—*Hydrocephalus hypersecretivus*. Since it has been conclusively proved by morphologic and histologic studies of the choroid plexus, by chemical analyses of the fluid, by a study of the effect of choroid extract on the secretion of cerebrospinal fluid, that the cerebrospinal fluid is the secretory product of the choroid gland, it would seem logical to suppose that a pathologic condition of the gland itself or a toxic substance in the fluid coming in contact with the plexus might bring about a hyperactivity of its cells. To establish this fact beyond the peradventure of a doubt will require the examination of many choroid plexuses and bacteriologic examination of the fluid in a large number of cases. I am now carrying on some studies in regard to the histology and pathology of the plexus which I trust will throw some light on the etiology of this type.

IV. With propriety we include in the varieties of hydrocephalus this fourth type, for which the term *Hydrocephalus occultus* has been chosen, which though paradoxical, is otherwise appropriate. The condition thus designated occurs usually in children, though occasionally in adults, and is characterized by excess of fluid in the ventricles, basal cysternae, and sometimes throughout the subarachnoid space, without necessarily any increase in the cranial dimensions. Symptomatically, this condition may be more closely allied to tumors, but from the point of view of treatment properly belongs to the problems of hydrocephalus in that the essential feature is an excessive accumulation of cerebrospinal fluid in the subarachnoid space. To illustrate this type, I may cite the following case:

A 3-year-old boy was referred to my service at the University Hospital, Oct. 17, 1911, by Dr. J. P. Crozer Griffith (File No. 10384). He was one of four children, two of whom had died in infancy. There was no evidence of injury at birth. September, 1911, the child fell down stairs, without apparent injury, but one week later developed convulsions and since that time has had seven or eight seizures. The child complained of headache a great deal, and had vomited frequently. A thorough physical examination failed to reveal any focal symptoms suggestive of intracranial tumor, with the exception of

the fact that the gait was a trifle unsteady, and in the movements of his arms as in feeding himself, there was some uncertainty or wavering. The latter might have been due to his defective vision. There was a papilledema of both disks, and vision was almost lost.

In the hope of saving at least a residuum of sight, pending the localization of a suspected tumor, a subtemporal decompression was performed. A considerable quantity of fluid was evacuated, and soon after the operation a large hernia cerebri developed, apparently under great tension. Evidently the hemisphere had been displaced by the distending ventricle.

It was not long before the papilledema subsided, revealing a well-marked, post papillitic atrophy. The convulsions ceased, the other evidences of intracranial tension disappeared, and but for the fact that vision remained impaired, the child's health was restored, and at an examination made three years later, the child was in perfect health.

An interesting feature in this case was the gradual subsidence of the hernia cerebri from one of large dimensions under terrific tension, to one of very moderate size. My interpretation of this striking change in the condition of intracranial tension was that the normal balance between the absorption and secretion of cerebrospinal fluid had been restored. The degree of optic atrophy indicated very positively that the duration of the primary lesion was very much longer than would be inferred from the history, that is, more than a matter of a few weeks. It was not until the intracranial tension had developed to the point of causing headache, vomiting and convulsions that the mother sought medical advice. Had the subtemporal decompression been performed before the stage of optic atrophy, the child's vision would have been preserved.

It will be seen at a glance that the proposed classification differs essentially from the venerable classification, internal and external hydrocephalus, and has a definite pathologic and physiologic basis. For practical consideration, therefore, it is now incumbent on the practitioner, before treatment is begun, to apply the tests which have been elaborated for distinguishing one from the other.

Test 1.—These tests[2] are based on the application to the cerebrospinal fluid of the phenolsulphonephthalein test for renal function. As the slight alkalinity of the commonly used solution is irritating to the brain and cord, a neutral solution is used. The solution is prepared in ampules containing 1 c.c. of the neutral solution. Under normal conditions, when the dye is injected into the lateral ventricle, it should appear in the fluid withdrawn by lumbar puncture within three to eight minutes. If therefore, after injection the fluid from the spinal canal is not stained within the specified time, it may be assumed that the drainage of the ventricles has been interrupted, and that we are dealing with hydrocephalus obstructivus.

Furthermore, it has been proved conclusively, first that the quantity of cerebrospinal fluid absorbed within the ventricles, if any, is a negligible quantity; and secondly, that from 30 per cent. to 60 per cent. of phenolsulphonephthalein should, under normal conditions, be secreted by the urine within the first two

2. These tests have been elaborated independently by Blackfan and Dandy and by Frazier and Peet, and the results are in entire accord.

hours. If, therefore, 1 c.c. is injected into the ventricle and the amount secreted by the first two-hour urine specimen estimated, we have at once additional evidence that we are dealing with the obstructive type.

The same test may be applied in the more unusual type of unilateral hydrocephalus. After the dye has disappeared from the urine following the test of one ventricle, the test may be applied to the other.

TEST 2.—From a lumbar puncture needle, 1 c.c. of cerebrospinal fluid is allowed to escape. A 2 c.c. record syringe, containing exactly 1 c.c. of the neutral phenolphthalein solution is attached to the lumbar puncture needle, and the piston withdrawn until the syringe is full. The solution of dye thus diluted is slowly injected into the subarachnoid space; the time of injection is noted and in five minutes a specimen is tested for the dye, and the entire amount of urine secreted in two hours collected. In the normal, a trace of the dye should appear in ten minutes and the entire amount excreted within the first two hours. Any marked diminution in the time or deviation from the amount indicates delayed absorption. In a case in which this test was recently applied, I saw the child when he was 16 months old in consultation with Dr. T. B. Holloway of Philadelphia. (History File No. 28358.) The delivery had been by forceps, the confinement lasted six hours, and after birth two lumps were noticed in the parietal region. Examination revealed the gross characteristics of hydrocephalus; the circumference of the head was 24 inches, and the anterior fontanel measured 2 by 4½ inches. The Wassermann reaction both on the spinal fluid and the blood was negative. The intraspinal pressure was 40 mm. of mercury. The child's mentality was not affected, except in so far that he had not begun to talk. He appeared intelligent and was well nourished.

June 30, 1915, a test for absorption from the subarachnoid space was made by Dr. M. M. Peet. Phenolphthalein appeared in the urine ten minutes after injection; within the two-hour period the total excretion was only 15 per cent. (delayed absorption). The test for ventricular drainage was made July 5, 1915. One cubic centimeter of the neutral phenolsulphonephthalein was injected into the ventricle. On lumbar puncture the dye did not appear until twenty minutes, and then only after the withdrawal of 120 c.c. of fluid. The fluid was withdrawn at five minute intervals, and twenty-five minutes after the injection, a slightly stronger trace appeared. The slowness with which the dye appeared in the cerebrospinal fluid, points to an obstruction, although not complete, of the ventricles. The fact that so large an amount of cerebrospinal fluid was found without the ventricles suggests that the obstruction was at the aqueduct of Sylvius, and that the excess of cerebrospinal fluid was secreted in the fourth ventricle. Evidently we were dealing with a hydrocephalus chiefly of the obstructive type, and to meet this condition I advised puncture of the corpus callosum. This was carried out by my assistant, Dr. L. H. Landon, July 6, 1915, and he made the observation at the time of the operation, that the fluid in the ventricles was still stained with the dye. This was rather interesting, in that it showed quite conclusively, that there must have been a very positive obstruction to the free circulation of the cerebrospinal fluid. The child was dismissed from the hospital in excellent condition, and thyroid feeding resumed.

If we are dealing with the internal or obstructive type, the absorption of phenolsulphonephthalein from the subarachnoid space and the excretion by the kidney is practically normal. If on the other hand, we are dealing with the nonabsorptive type, the time of appearance of the dye in the urine is delayed and it may not appear for an hour or more, and the amount secreted in the two hour period is correspondingly low; frequently but a trace is detected. In a few cases no phenolsulphonephthalein reaches the urine in four or six hours.

SUMMARY OF CLINICAL TESTS

First examination:

1. Lumbar puncture.
2. Withdrawal of 1 c.c. of cerebrospinal fluid.
3. Attach 2 c.c. record syringe filled with 1 c.c. neutral solution of dye.
4. Withdraw piston until syringe is full.
5. Inject solution slowly into lumbar subarachnoid space.
6. Withdraw needle.
7. Test urine for phenolsulphonephthalein every 5 minutes until dye is detected.
8. Estimate total amount of dye excreted in the first two-hour specimen of urine.

Second examination (following day, or after dye is no longer found in urine):

1. Puncture of the lateral ventricle.
2. Inject 1 c.c. neutral phenolsulphonephthalein solution.
3. Lumbar puncture. Examine for dye every five minutes until dye appears.
4. Test five-minute specimens of urine.
5. Estimate total amount of dye excreted in first two-hour specimen.
6. In calculations, the amount of dye lost by lumbar puncture must be taken into consideration.

TREATMENT

It is not the purpose of this paper to enter into any detailed discussion of the treatment of hydrocephalus. To do so in extenso would necessitate opening the pages of medical history at the time when Hippocrates boldly tapped the hydrocephalic ventricle, and carrying the reader on numberless surgical flights and falls. The number of operations that have been devised to grapple with this Charybdis of surgical endeavor are conspicuous more for their ingenuity than for their life-saving accomplishment. To record here the mortality of many operations for hydrocephalus would be too depressing and would dampen the ardor of those seeking to shed new light.

An attempt will be made merely to suggest methods of treatment, and in doing so to keep within the bounds of reasonable risk and promising results.

I realize, of course, that the excessive accumulation of fluid in the hydrocephalic may not be the only pathologic factor, and that the ability to cope with this may not be the key to final and complete restoration of health. At the same time, it is reasonably certain that the excessive accumulation of cerebrospinal fluid is the dominant disturbing element of this disease, and must be given consideration.

The suggestions as to treatment will be made to conform with our classification, on the ground that different etiologic factors indicate differing therapeutic methods. The simplest and most effective method of dealing with hydrocephalus obstructivus is puncture of the corpus callosum, the *Balkenstich* of Anton and Bramann. This operation is in hydrocephalus devoid of difficulty, and in the simplest way provides an outlet for the pent-up fluid into the subarachnoid space, where

it may be absorbed. Because of its simplicity and effectiveness, it should supplant all methods of ventricular drainage; drainage by gold or platinum tubes, drainage into the longitudinal sinus, drainage into the jugular veins, drainage of the fourth ventricle, etc.

In the nonabsorptive type, greater technical difficulties are encountered. Speaking now with some reservation, because my technic for this is in the development stage, I venture to recommend the establishment of a drainage tract into the pleural cavity. The distance is not too great, the anatomic features are adaptable, and the pleural cavity is a convenient and readily accessible reservoir.

When the lesion is due to hypersecretion I resort to thyroid feeding. In the numerous experiments which we made on the activity of the choroid plexus, as a secretory organ, at once the most conspicuous and impressive finding was the influence of thyroid feeding. Subjected to every possible test in the elimination of sources of error, it was found that thyroid invariably acted as a depressor on the choroid plexus, and invariably reduced the secretion of cerebrospinal fluid. This reduction was notable in amount, in constancy and in duration. In some instances the experiments covered periods of five consecutive hours. With this evidence of what appears to be an almost specific influence of thyroid extract on the choroid plexus—as it were, an interglandular relation—I began to apply the treatment in appropriate cases. To the pediatrist, who is brought in contact with these cases in much larger numbers than the surgeon, I hope this line of treatment may appeal sufficiently to give it a thorough trial, and, by the results, to establish or disestablish it as an approved method. In the case of a baby which I saw June 13, 1914, in consultation with Dr. G. R. Anderson, thyroid feeding was inaugurated. The baby, at that time 5 weeks old, was the first child of apparently healthy parents. Labor was not instrumental, but the head was engaged in the birth canal over an hour. At the time of birth the child seemed in every way to be normal, and it was not until two weeks after birth that the doctor's and parents' attention was called to the fact that the size of the head had increased disproportionately to the natural growth. At the time of my examination, the child appeared thin and poorly developed. There were no other congenital defects. The head was unquestionably large, larger than it should have been for a child of that age and size, the forehead prominent and unusually large. The circumference of the head was 16⅝ inches, and from a point in front of the external auditory meatus on one side, to a similar point on the other, measured 12¼ inches. The question of operation, of course, never came up for consideration. I was constrained to give an unfavorable prognosis, in view of the fact that the child's head had increased in size so rapidly

in the first few weeks of its existence. Based entirely on our observations in the laboratory, I advised the administration of thyroid extract beginning in doses of 1/6 gr. and gradually increasing the dose up to the point of tolerance.

July, 1915, the physician attending reported that the fontanels had closed, that there had been no disproportional increase in the circumference of the child's head, that the child had begun to talk and walk with assistance, and that apart from an internal squint, the child seemed perfectly well and normal. Whatever may have been the influential factor, the disease had been arrested.

This brief sketch of the causes and treatment of hydrocephalus it is hoped will stimulate physicians to take up anew this old vexing problem, to subject their cases to a more critical analysis for the differentiation of one type from another, and to be influenced in management and treatment by the more recent contributions to our knowledge of this disease, not losing sight of the fact that experimental evidence points very convincingly to a retarding influence of thyroid extract on the secretory activity of the choroid plexus.

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METABOLISM STUDIES IN HEMOPHILIA*

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Since the year 1820 when Nasse of the University of Bonn formulated the law that males alone are the bleeders, and the disease, hemophilia, is transmitted by normal females through their marriage with normal males, much has been written on the subject of hemophilia. This disease, which Grandidier termed "The most hereditary of hereditary diseases," has been discussed from all angles and points of view, and the only conclusion that has been reached is that the coagulation of the blood was defective (Wright, Addis, et al.).

What the cause of this delayed coagulation is has been explained in various ways. Sahli and Morawitz have stated that the calcium content of the blood is normal in this disease; that the thrombogen content of the blood is normal; and they ascribe the delayed coagulation to a deficiency in the thrombokinase (the activator of the thrombogen) of the blood. This theory has been contradicted by Addis, who believes that the fault lies in the composition of the thrombogen, which (though normal in quantity) is so changed in character that it liberates the thrombin with difficulty.

According to Weil, the delayed clotting is due to an excess of antithrombin, whereas, according to Howell the deficiency of the blood in thrombogen is to be blamed for the defect. It was Wright who ascribed the delayed coagulation of the blood in hemophilic subjects to a lack of calcium.

In examining the literature on this subject I have failed to find any records of metabolism studies in this condition. Since I have had occasion to make such studies of two cases of hemophilia, I think it will be interesting to report my results.[1]

The two cases studied were of different types of hemophilia. They were both in boys. Julius was about 9 years old, and gave a typical history of hereditary hemophilia. The boy Alter, was about 6 years old and was not a hereditary "bleeder." Dr. Hess thus presents the clinical history of this boy (Alter):

> The child has been under our observation for the past three years. . . . Attention was first directed to him on account of his tendency to bleed from

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1. The clinical studies of these cases were reported by Dr. A. F. Hess, Johns Hopkins Hosp. Bull., 1915, xxvi, 372. A preliminary communication was made by A. F. Hess and Max Kahn, Biochem. Bull., 1915, iv, 213.

minor cuts, to manifest frequent "black-and-blue marks" and to have severe hemorrhages from the nose. As far as can be ascertained, he is the sole member of his family who is a bleeder, so that we must consider him as affording an instance of the sporadic type of hemophilia. He is fairly well nourished, weighs about 50 pounds and shows no other abnormality except a moderate degree of ichthyosis of the skin.

In both of the cases it was found that there was delayed coagulation of the blood. In the case of Julius the blood clotted in about eighteen minutes, and in the case of Alter it clotted in thirty-four minutes. On analyzing the blood of these patients for calcium and comparing the results with normal cases we obtained the results shown in Table 1:

TABLE 1.—Calcium Oxid in Blood

Normal Controls, Gm.	Alter, Gm.	Julius, Gm.
0.072	0.047	0.068
0.068	0.056	0.069
0.074	0.058	.....
0.066	0.058	.....
0.071	0.062*	.....
0.073	0.064*	.....
0.062	.....	.....
0.068	.....	.....
0.075	.....	.....
0.069	.....	.....
0.075	.....	.....
0.065	.....	.....

* Calcium lactate (2 gm.) had been given for the previous three days by mouth.

It will be seen that in the case of Julius, atypical hemophilic, the lime in the blood was normal, whereas in the case of Alter the calcium content of the blood was decreased.

We reasoned that if the calcium deficiency was the sole cause of the failure of the blood to clot in the case of the boy Alter, this defect could be remedied by adding to the blood the missing lime salt. We, therefore, prepared a solution of calcium chlorid in physiologic saline (0.04 gm. Ca calculated from $CaCl_2$ in 1,000 gm. saline) of such strength that the addition of a drop of this solution to ten drops of blood will approximately supply the deficiency in calcium. (Normal CaO in blood calculated to be 0.08 parts per thousand). Table 2[2] shows the effect of the addition of the calcium solution on the clotting

2. Hess, A. F.: Johns Hopkins Hosp. Bull., 1915, xxvi, 373.

TABLE 2.—Effect on Clotting Time of Blood of Addition of Calcium Chlorid Solution

A. Normal Case

Clotting Time	Control	Drops of Ca Solution added			
		1	2	3	4
4 min.	+	.....	+	+	+
5 min.	++	.....	++	++	+
6 min.	+++	.....	+++	++	++
7 min.	.....	.....	.....	++	++
9 min.	.....	.....	.....	++	++
11 min.	.....	.....	.....	++	++
13 min.	.....	.....	.....	++	++
15 min.	.....	.....	.....	+++	++
17 min.	.....	.....	.....	.....	++

B. Hemophilia Calcipriva (Alter).

5 min.	—	—	—	—	—
9 min.	—	—	—	—	—
11 min.	—	+	++	—	—
13 min.	—	+	+++	+	—
17 min.	—	+++	.....	++	+
21 min.	—	.....	.....	++	++
26 min.	+	.....	.....	+++	+++
34 min.	+++	.....	.....	.....	.....

C. Typical Hemophilia

4 min.	—	—	—	—	—
8 min.	—	—	--	—	—
10 min.	+	++	+	++	+
14 min.	++	++	++	++	++
18 min.	+++	++	++	++	++
22 min.	.....	++	++	++	++
26 min.	.....	++	+++	++	++
30 min.	.....	.....	.....	+++	++

+++ denotes complete clotting time.

time of the blood. The determinations of the coagulation time of the blood were made by Dr. A. F. Hess. This table is taken from Dr. Hess' report.

It will be observed that in the normal case the addition of two drops of the lime solution had no effect, and the addition of three and four drops delayed the coagulation. In the case of Alter the addition of one drop quickened coagulation from thirty-four minutes to seventeen minutes; the addition of two drops caused clotting in thirteen minutes; the addition of three and four drops again slowed coagulation to twenty-six minutes. In the case of Julius the addition of calcium chlorid slowed the coagulation of the blood.

Having observed these effects we thought it advisable to study the general metabolism of the two hemophilic children, and compare them with normal children. The following procedure was adhered to in the study of the metabolism.

Both patients were fed on a diet of milk, eggs, butter and bread. Duplicate samples of each substance given the patients were sent daily to the laboratory where they were weighed and analyzed. The urine and the feces were collected daily for examination.

The experiment may be divided into three periods: 1. A fore-period, of a duration of three days, during which time the patients were kept in bed and fed the diet above mentioned, without being given any medication. 2. A calcium-feeding period also lasting for three days, from August 19 to 22, 1914 (during this time the patients received daily 2 gm. of calcium chlorid and 1 gm. of magnesium oxid). 3. An after-period, lasting three days, during which time there was given no medication.

EXPERIMENTAL METHODS

In the analysis of the food the following methods were employed: Nitrogen by the Kjeldahl method; total sulphur by the Wolff and Osterberg method, a modification of the Benedict method; calcium and magnesium by the McCrudden method, after ashing and extracting the ash with hydrochloric acid; phosphorus by the Neumann method.

In the urinary analyses, in addition, ammonia was determined by the Folin method, urea by the Benedict method, and total and ethereal sulphates by the Folin method. The inorganic sulphates were computed by subtracting the ethereal sulphates from the total sulphates, and the neutral sulphur by subtracting the total sulphate sulphur from the total sulphur. Total phosphorus was determined by the Neumann method, the phosphorus being weighed as magnesium pyrophosphate; creatin and creatinin by the Folin method, and uric acid by the Krieger-Schmidt method.

For the feces the same methods were used. In the case of the fat the Kumagawa and Suto method was employed. The results of the examinations are summarized in the four accompanying tables (Tables 3, 4, 5 and 6). These show that the disturbances of nutrition are not at all similar in the two children. In order to appreciate the results we have obtained it is wise to compare them with the metabolism of normal children, and we take the case of Herman S., a boy 5 years of age, as a standard (Tables 7 and 8).

TABLE 3.—Summary of Metabolism During the Three Periods

A. TYPICAL HEMOPHILIA (JULIUS)

Substance	Fore Period (Average per Diem)				Feeding Period (Average per Diem)				After Period (Average per Diem)			
	Food	Urine	Feces	Bal.	Food	Urine	Feces	Bal.	Food	Urine	Feces	Bal.
Nitrogen.....	16.14	10.55	3.75	+1.84	16.10	10.13	2.28	+3.69	14.61	10.38	1.93	+2.30
Sulphur.....	1.90	0.98	0.87	+0.05	1.93	0 91	0.95	+0.07	2.06	0.91	0.95	+0.20
Phosphorus..	3.78	2 13		+1.65	3.26	3.03		+0.23	3.79	2.51		+1.28
Chlorin.....	7.94	6.74		+1.20	9.19	8.17		+1.02	8.58	6.78		+1.80
Calcium.....	1.59	0.206	0.824	+0.56	3.39	0.466	2.08	+0 85	1.47	0.17	0.71	+0.59
Magnesium...	0.51	0.177	0.133	+0.20	1.64	0.84	0.61	+0.19	1.45	0.70	0.53	+0.22

B. HEMOPHILIA CALCIPRIVA (ALTER)

Nitrogen.....	11 53	6.75	2.07	+2.71	11.50	6.99	2.17	+2.54	11.30	7.26	2.78	+1.26
Sulphur.....	1.31	0.88	0.30	+0.13	1.94	1.02	0.33	+0.59	1.84	0.95	0.29	+0.60
Phosphorus..	3.49	2.98		+0.51	3.36	3.09		+0.27	3.91	3.55		+0.36
Chlorin.....	4.60	4.09		+0.51	6.00	5.06		+0.94	5.03	3.44		+1.59
Calcium.....	1.37	0.39	1.80	—0.82	3.60	0.73	2.24	+0.63	1.33	0.21	0.81	+0.31
Magnesium...	0.42	0.19	0.11	+0.12	1.56	0.67	0.32	+0.57	0.67	0.14	0.07	+0.46

TABLE 4.—Calcium and Magnesium Metabolism (Alter)

Day	Intake	Urine	Feces	Balance	Intake	Urine	Feces	Balance
1.....	1.35	0.37	1.73	—0.75	0.35	0.14	0.09	+0.12
2.....	1.40	0.43	1.94	—0.97	0.47	0.21	0.12	+0.14
3.....	1.37	0.39	1.71	—0.72	0.45	0.21	0.14	+0.10
4.....	3.55	0.72	2.31	+0.52	1.92	1.21	0.56	+0.15
5.....	3.62	0.73	2.14	+0.75	1.35	0.42	0.21	+0.72
6.....	3.65	0.75	2.28	+0.62	1.41	0.37	0.20	+0.84
7.....	1.32	0.12	0.46	+0.74	0.87	0.16	0.09	+0.62
8.....	0.27	0.24	0.88	+0.15	0.62	0.10	0.05	+0.47
9.....	1.42	0.27	1.11	+0.04	0.54	0.17	0.09	+0.28

During Period 2, calcium and magnesium were added to the food.

TABLE 5.—URINARY NITROGEN PARTITION

A. HEMOPHILIA CALCIPRIVA (ALTER)

Day	Amount, c.c.	Total N, gm.	Urea N, Per Cent. of Total N.	Ammonia N, Per Cent. of Total N.	Creatinin N, Per Cent. of Total N.	Uric Acid N, Per Cent. of Total N.
1	750	10.56	82.7	4.2	3.1	2.25
2	720	9.87	83.4	4.4	3.4	1.98
3	810	11.23	82.5	3.9	2.9	2.72
4	690	10.07	82.9	3.9	3.2	2.15
5	760	10.11	82.7	4.1	3.1	2.22
6	770	10.27	83.2	4.3	3.2	2.14
7	840	9.87	83.5	3.8	3.4	2.07
8	720	10.56	81.9	4.4	3.3	2.24
9	730	10.72	82.8	4.2	3.4	2.05

B. TYPICAL HEMOPHILIA (JULIUS)

1	450	6.78	82.7	4.2	3.5	1.2
2	530	7.18	83.8	4.4	3.4	1.4
3	470	6.27	83.6	3.9	3.7	1.4
4	490	6.86	85.2	4.3	3.4	1.3
5	520	7.02	82.9	4.4	3.1	1.5
6	580	7.10	84.4	4.1	3.6	1.2
7	620	.....	....	...	...	...
8	490	.....	....	...	...	...
9	510	7.26	82.8	4.2	3.5	1.2

TABLE 6.—Sulphur Output in Urine and Feces

A. HEMOPHILIA CALCIPRIVA (ALTER)

Day	Urine				Feces, Total S, Gm.
	Total S, Gm.	Ethereal SO_4 Per Cent. of Total S	Inorganic SO_4 Per Cent. of Total S	Neutral S Per Cent. of Total S	
1	0.82	12.6	72.9	14.5	0.27
2	0.97	14.2	70.1	15.7	0.32
3	0.85	13.7	72.0	14.3	0.31
4	0.89	12.9	69.9	17.2	0.38
5	1.02	12.4	75.0	12.6	0.29
6	1.15	12.2	74.7	13.1	0.33
7	....	....	....	....	0.27
8	....	....	....	....	0.28
9	0.95	13.4	71.1	15.5	0.31
Average	0.95	13.0	72.2	14.7	0.31

B. TYPICAL HEMOPHILIA (JULIUS)

1	0.87	14.3	68.1	17.6	....
2	0.92	14.5	69.2	16.3	....
3	1.05	16.2	65.7	18.1	0.87
4	0.97	12.9	67.9	19.2	0.98
5	0.86	16.1	68.4	18.5	0.85
6	0.89	14.4	68.0	17.6	1.04
7	0.87	14.7	69.0	16.3	0.95
8	0.91	13.8	70.6	18.6	0.90
9	0.94	13.2	69.2	17.6	1.02
Average	0.93	14.3	68.3	17.3	0.94

TABLE 7.—Nitrogen Metabolism—Normal Boy—5 Years Old (After Schwarz)[3]

Nitrogen in food	9.567 gm.
Nitrogen in urine	7.485 gm.
Nitrogen in feces	0.696 gm.
Retention	14.5 per cent.
Absorption	92.7 per cent.

3. Schwarz, H.: Jahrb. f. Kinderh., 1910, lxxii, 549.

TABLE 8.—SULPHUR METABOLISM—NORMAL BOY—5 YEARS OLD (AFTER SCHWARZ)[3]

Total sulphur in food	0.968 gm.
Sulphur in urine	0.592 gm.
Sulphur in feces	0.075 gm.
Retention	31.1 per cent.
Absorption	92.2 per cent.

TABLE 9.—(MCCRUDDEN AND FALES.)[4] NORMAL BOY, W. McC.
Weight at beginning of experiment, 27 kg.; weight at end, 27.2 kg.

URINE

Day	Nitrogen	Sulphur	Oxid	Magnesium Oxid	Phosphorus Pentoxid
1	10.43	0.729	0.374	0.153	2.34
2	10.65	0.744	0.406	0.139	2.22
3	10.35	0.717	0.373	0.154	2.23
4	11.07	0.832	0.348	0.152	2.06
5	11.35	0.808	0.332	0.157	2.58
6	11.12	0.810	0.390	0.154	2.68
Total	64.97	4.640	2.223	0.909	14.11

FECES

	Dry Feces					
1	2.28	1.232	0.133	1.518	0.267	0.686
2	19.0	0 997	0.085	1.846	0.263	0.718
3	20.9	1.165	0.115	1.784	0.288	0.768
4	23.5	1.173	0.113	2.084	0.301	0.839
5	15.3	0.811	0.063	1.318	0.220	0.526
6	21.1	1.110	0.073	1.643	0.279	0.856
Total	122.6	6.488	0.582	10.193	1.618	4.393

Total excretion	71.46	5.222	12.42	2.53	18.50
Total in food	86.10	6.138	14.988	3.263	24.709
Retention	14.64	0.916	2.57	0.73	6.20
Per cent. retained	17.0	14.9	17.1	22.4	25.0

4. McCrudden, F. H., and Fales, H. L.: Jour. Exper. Med., 1912, xv, 453.

The protein metabolism as measured by the nitrogen and sulphur output, in the case of the two hemophilic children, is similar to the nitrogen and sulphur metabolism in the 5-year-old boy studied by Schwartz. The absorption, retention and excretion are about the same, and the partition of the nitrogen and the sulphur into the various fractions gave normal average results.

The results of the study of the mineral metabolism should be compared with the records of the study by McCrudden and Fales[4] of the metabolism of a boy weighing 27.3 kg.

Compared with the normal boy, the excretion of calcium in the urine as compared with the excretion of calcium in the feces was about the same in both the hemophilic patients. In the case of Alter—the case of hemophilia calcipriva—there was a negative calcium balance in the fore-period, a positive calcium balance in the line-feeding period, and a decreasingly positive calcium balance in the after-period. The mineral metabolism in the true hemophilic, Julius, appears normal.

From these observations it would appear that not all hemophilia patients present similar pathologic-chemical disturbances. There seems to be no derangement in the metabolism as measured by the intake and output of nitrogen, sulphur, calcium, etc., in the case of hemophilia vera. There are, however, certain bleeders in whom the disturbing factor seems to be a lack of calcium content of the blood, and an inability on the part of their organisms to assimilate properly the lime from the food. In these cases the remedy indicated would be to administer the lacking mineral constituent in the form of the chlorid or the lactate of calcium.[5]

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5. In addition to the previous numbered references, the following may be consulted:

Pratt, J. H.: Osler and McCrae, Modern Medicine, 1915, iv, 717.

Bulloch and Fildes: Treasury of Human Inheritance, London, 1911, xiva, 169.

CARMIN TEST FOR THE DURATION OF THE COMPLETE FOOD PASSAGE IN INFANTS AND CHILDREN

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During the last half century many new methods of investigation have been employed in gastro-intestinal diagnosis. A variety of test meals has been devised to determine the digestibility of foods.

Carmin, because it stains the ingesta red, and charcoal, which passes the alimentary canal unchanged, have been used to mark off stools from one diet to another. Bismuth or barium is used in roentgenography to enable us to watch the actual progress of a test meal, and as a result, various lesions of the digestive tract have been diagnosticated.

However, owing to their tender age, young children cannot profit by these advances in the same degree as adults. This fact, in all probability, explains why the diagnosis of gastro-intestinal diseases in childhood is less far advanced than in adults.

The use of carmin in gastro-intestinal diagnosis dates back to the early seventies, but it is only quite recently that systematic reports of work done in this field have been published. The first to make such reports have been French pediatrists, to wit, Triboulet,[1] and Nobécourt and Merklen,[2] respectively.

These men have been closely followed, and without knowledge of their work, by Spivak,[3] an American.

Triboulet used the carmin test on fifty children, but few of whom were less than 2 months old. Some infants were healthy, while others had gastro-enteric disorders. Carmin was given in solution. The author seems to have been interested chiefly in the duration of the total food passage. In healthy children, breast or bottle fed, at least sixteen hours were required for total elimination, and the limits ranged from sixteen to twenty-two hours. The author is not so much interested in the first appearance of carmin in the stools, and only states that the briefest interval noted in healthy children was ten hours. His chief contention is that a complete passage of less than fourteen hours is abnormal. In children with accidental catarrh, the passage time varied

1. Triboulet: Bull. Soc. de pediat., Paris, 1909, xi, 512; 1910, xii, 85.
2. Nobécourt and Merklen: Bull. Soc. de pediat., Paris, 1910, xii, 4.
3. Spivak: Denver Med. Times, 1910, xxx, 171.

from nine to twelve hours, and in hyperemic and ulcerated forms from three to eight hours. The main point deduced is that (since the more severe the condition the higher up the lesion) these quick passages have a certain diagnostic value.

Nobécourt and Merklen in taking up the subject tested only healthy infants under 3 months of age (five under 1 month). Ten observations were made on six children. The first carmin colored stools came away at all periods from three to twenty-one hours, the average being ten hours. In seven observations the interval was from three to ten hours. The last colored stools came away in from eleven to thirty-eight hours —average twenty hours. The complete passage time in eight observations was not over twenty-one hours.

The chief difference between the two sets of observations refers to the first appearance of the carmin in healthy children. It was much earlier in Nobécourt and Merklen's cases, but the children were considerably younger.

Spivak applied the test to nineteen children, the ages of whom were from 4 days to 3 months. The first appearance of carmin was from three to ten hours, agreeing exactly with the figures of Nobécourt and Merklen. The maximum passage time was twenty-one hours, again agreeing with the latter figures.

Spivak also states that since in adults the feces in the colon represent the accumulation of the previous forty-eight to thirty-eight hours, i. e., of the three meals eaten on the second day before defecation, the feces must travel at a much slower rate than carmin, which may appear in the stools of healthy subjects five hours after it is swallowed, this being the shortest interval found by him in examining a large number of adults. Hence the carmin must diffuse itself directly to the colon and stain feces already present therein. This must to a certain extent be true of children.

Recently I have tested the time of the food passage on two separate sets of subjects. The first (Table 1) comprises twenty-one very young and healthy breast fed nurslings from 1 to 6 days old, at the Jewish Maternity Hospital. Carmin was given in powder form in ½ grain doses. These babies were nursed every three hours (two with subnormal temperature, nursed every two hours, took very little milk). The temperature varied from 96 to 99 F. The number of stools daily was two or three. The time of the appearance of red stools varied from four hours to eighteen hours, and for the disappearance of the stain from four to twenty hours were required. While my material was all newly born children, these figures do not differ radically from those of Nobécourt and Merklen and Spivak.

TABLE 1.—FROM THE CHILDREN'S SERVICE AT THE JEWISH MATERNITY HOSPITAL

Name	Sex†	Age of Child	Breast-fed How Often	Appearance of Red Stools*
		Days Hrs.	Hrs.	Hrs. Mins.
S.	♂	4 22	3	9 10
W.	♂	3 11	2	9 10
G. L.	♂	3 13	3	7
Wein.	♂	6 2½	3	12 20
B.	♂	6 2½	3	12
V.	♀	2 6	2 and 3	4 30
W.	♀	2 6	3	6 30
F.	♀	1 3	3	18
Vig.	♀	1 2	3	12
Alt.	♀	4 3	2	7 30
S.	♀	4 12	3	8 30
B. A.	♀	3 2	3	9
G.	♂	2 4	3	7
D.	♂	1 10	3	10
L.	♂	2 5	3	10½
C.	♀	4 1	3	8
R.	♀	3 2	2	9½
K.	♀	2 6	3	7½
Ra.	♂	3	3	9½
Tl.	♂	3 6	3	8½
Oel.	♀	2 5	3	7

* The stain of the stools persisted from four to twenty hours.
† ♂ means male; ♀ means female.

My second material came (Table 2) from the Beth Israel Hospital —twenty-five cases of sick children, the ages varying from 6 weeks to 6 years. The dose of carmin given was from 1 to 2 grains. The children in the great majority of cases had subfebrile temperatures (under 101 F.) and a number had been attacked with severe maladies like bronchopneumonia, endocarditis, etc. The shortest first appearance was five and a half hours, the longest fifty-two hours.

The average first time for the appearance of the carmin was from twenty-five to thirty hours. The passage time was not recorded.

In summing up we may quote also some old figures in regard to carmin passage cited by Triboulet, without names or references.

HEALTHY CHILDREN

Anonymous (Triboulet) First appearance of carmin in healthy infants, average ..... 3 hrs. 50 min.
Nobécourt and Merklen (7 in 10 cases) ..... from 3 to 10 hrs.
Spivak ..... from 3 to 10 hrs.
Hymanson ..... from 4 to 18 hrs.

These figures show some congruence. Triboulet saw no carmin before ten hours.

DURATION OF ENTIRE PASSAGE

Anonymous, average ..... 9 hrs.
Triboulet ..... 16 to 20 hrs.
Nobécourt and Merklen ..... Under 21 hrs.
Spivak ..... Not over 21 hrs.
Hymanson ..... 4 to 20 hrs. (average 12 hrs.)

These figures show considerable agreement.

TABLE 2.—FROM THE CHILDREN'S SERVICE AT THE BETH ISRAEL HOSPITAL

Name	Age	Diagnosis	Temperature, F.	Food	Appearance of Red Stools, Hrs.
S. Tr.	1 1/12	Pneumonia	100	Breast milk and barley water	22
S. B.	1⅛	Bronchopneumonia; convalescing	Normal		8
H. P.	1⅝	Empyema	100 to 102	Third diet	28½
T. L.	1½	Pneumonia; convalescing	Normal	Toast, cereals and milk	31
A. M.	2	Chronic empyema	Normal	Soft diet	5½
S. K.	4	Lobar pneumonia; convalescent; constipation	Normal	Third diet	52
M. S.	5	Rheumatic endocarditis	100.5	Cereals, toast, eggs, milk and fruit	18½
F. W.	6	Endocarditis	100	Cereals, toast, eggs, milk and fruit	24½
R. A.	6	Empyema	99 to 102.5	Soft diet	23¾
E. B.	4½	Chronic empyema; constipation	Normal	Soft diet	49½
S. Kr.	1¼	Lobar pneumonia	100	Thirds, toast and cereals, also orange juice	28
M. M.	2	Intestinal intoxication	100	Milk ⅔; barley water ⅓; lactose 3%	31
B. C.	⅔	Bronchopneumonia	100	Breast milk and some barley water	12½
F. Z.	½	Spasmophilia	Normal	Milk ⅔; barley water ⅓; sugar 3%	20¼
J. B.	7/12	Furunculosis	Normal	Milk ⅔; barley water ⅓; lactose 3%; Mellin's food	11
I. W.	6*	Normal baby	Normal	Breast fed; also milk and barley water; sugar 2%	7
M. G.	⅝	Lobar pneumonia	100 to 101	Breast milk and barley water and lactose 3%	45
J. L.	9½†	Enteritis	100.5	Elweissmilch and barley water; also 2% of lactose	36
J. M.	⅝	Convalescing from bronchopneumonia	100	Breast fed	10
B. D.	8½†	Convalescing from ear trouble	Normal	Breast fed; milk ⅔, barley water ⅓	8
I. G.	5½†	Diarrhea	100.5	Elweissmilch	12
B. G.	29‡	Pneumonia	100	Breast fed	28
S. R.	½	Gastro-enteritis	97 to 101	Equal parts of milk and barley water; sugar 3%	21
R. L.	11/12	Bronchitis	101	Milk ⅔; barley water ⅓; sugar 4%	11
M. T.	¾	Abscess of scrotum	100	Breast fed, also equal parts of milk and barley water	13

* Weeks; † Months; ‡ Days.

SICK CHILDREN

The marked differences between the small figures of Triboulet (three to twelve hours) for complete passage, and the large figures of the author (average twenty-five to thirty hours), seem to be wholly due to the fact that Triboulet's sick infants all had diarrhea or enteritis, while in the author's material, bowel troubles were in a minority.

In closing we may call attention to the suggestion of Nobécourt and Merklen that the carmin test should prove of value in benign stenosis of the pylorus. If carmin given by the mouth does not appear in the stools within a definite interval, there might be good reason to suspect the existence of pyloric obstruction. In seven of ten observations the longest deferred first appearance was ten hours after ingestion, although in the three others, this was prolonged up to twenty-one hours.

We may also mention Dr. Seymour Basch's[4] conclusions in a recently published paper on the use of the carmin test in adults. He speaks of it as a simple, harmless, reliable and convenient method for the demarkation of stools; for the estimation of gastro-enteric motility and patency; for the detection of fistulous communications of the alimentary canal with the exterior or other hollow organs; for the location of the distal end of a duodenal tube and to aid in the differentiation between esophageal diverticulum and dilatation.

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4. Basch, S.: Jour. Am. Med. Assn., 1913, lxi, 1295.

PROGRESS IN PEDIATRICS

RESUME OF WORK ON TUBERCULOSIS IN CHILDREN FOR 1915 *

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MODES OF INFECTION

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2. Grulee, C. G., and Harms, F.: Tuberculosis as a Disease of the New-Born, Am. Jour. Dis. Child., 1915, ix, 322.
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7. Frisch, A.: Pathogenesis of Tuberculosis in Childhood, Prag. med. Wchnschr., 1915, xl, 201.
8. Bartlett, F. H.: The Pathology of Tuberculosis in Childhood, Arch. Pediat., 1915, xxxii, 536.
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W. H. Park,[1] discussing the transmission of tuberculosis in childhood, says that intrauterine infection occurs occasionally but accounts for a very small proportion of the tuberculosis of childhood. In most of these cases the mother has had extensive miliary tuberculosis. There seems no evidence that intrauterine infection ever proceeds from the father. After birth the human infant is endangered not only by bacilli from human sources, but from diseased cattle. Cows contaminate the milk extensively, only when they have disease of the udder. One or two cows with seriously diseased udders are capable of dangerously infecting the mixed milk of a whole herd. It is fairly certain, however, that when tubercle bacilli contained in milk are heated to 140 F. and held at that temperature for twenty minutes they are destroyed. Butter is known to contain tubercle bacilli when made from milk infected with them. The evidence that butter actually transmits tuberculosis is less direct than that milk does. This is partly because the bacilli are fewer,

* Submitted for publication Jan. 10, 1916.

and because butter is not usually taken at an age when tuberculosis of the bovine type is most likely to be transmitted.

Although recent investigations have definitely proved that bovine bacilli infect infants and to some extent older children, yet the evidence is equally clear that most infections are transmitted by one human being to another. In the case of infants and young children, the greatest danger lies in being attended by a tuberculous mother or nurse. This direct human contamination can be avoided only by keeping the child from contact with tuberculous persons.

C. G. Grulee and F. Harms[2] call attention to the subject of tuberculosis in the new-born, with the hope not of adding a great deal to the literature, but of stimulating interest in the subject, which has been so long neglected.

Their case is as follows: The child was born of a mother with a supposedly healed tuberculosis, and with only one subjective symptom, a severe leukorrhea of unknown cause. The mother survived the child many months. The baby fifteen hours after birth developed a high temperature, which continued until his death on the eleventh day. There was a distinct tendency to regurgitation of food. Generalized convulsions developed on the seventh day and continued up to twenty-four hours before death. On physical examination the child showed only an enlarged liver and spleen. At necropsy a generalized tuberculosis, affecting most markedly the abdominal organs, and especially the periportal lymph glands, liver, and spleen, was found. The tuberculosis was miliary in type but the stage of the tubercles suggested that the disease had begun in utero. Many tubercle bacilli were found in all sections of the tuberculous area.

The authors find that congenital and placental tuberculosis is being reported with increasing frequency within the last few years. From a pathologic standpoint there are two forms of tuberculosis of the new-born, both miliary in type, namely the pulmonary and the abdominal. The pulmonary form is not always of placental origin and seems much more likely to result from aspiration of the liquor amnii or vaginal secretions at the time of birth. The abdominal form may be termed the general miliary form, for, while the lesions are more marked in the abdominal cavity, the thoracic viscera are always involved.

Clinical data of tuberculosis of the new-born are so scant that no conclusions can be drawn as to the symptomatology. The writers think it not at all impossible that the early temperature in their case had nothing to do with the tuberculosis, but was simply the so-called inanition fever, frequently noted at this time. The combination of enlargement of the spleen, high, irregular temperature, and enlargement of the liver, together with tuberculosis in the mother is suggestive.

In diagnosis little can be expected from the tuberculin tests. In the newborn the tuberculin test has been negative almost without exception. The prognosis has always been fatal. The duration of life has varied from twenty-four hours to twenty-five days.

M. Péhu and J. Chalier[3] have collected from the literature sixteen undoubted cases of congenital tuberculosis, which were not included in their last paper on the subject or which since have been published. They conclude that authentic cases of congenital tuberculosis are rare, as they were able to collect only fifty-one, all told. The infection occurs during or at the end of pregnancy, when the placental circulation is established, and is the result of a bacillemia which is usually a terminal event. They should always be regarded as examples of transplacental heredo-contagion.

M. Péhu and J. Chalier[4] summarize their paper on the inheritance of tuberculosis as follows: Direct transmission of tuberculosis by the sexual cells (ovule and spermatozoa) has not been demonstrated. Theoretically it is possible, but in the strict sense of the word, heredity of the germ of tuberculosis does not exist. Transplacental herdo-contagion exists, but owing to its extreme rarity this mode of propagation of tuberculosis is of little importance. The dystrophies which at times have been observed in tuberculous children are not specific in character. It has not been proved that there is a special hereditary predisposition to tuberculosis. The descendants of the tuberculous offer a receptive soil for all diseases and not for tuberculosis in particular. Nor has it been demonstrated that there exists an hereditary transmission of immunity for tuberculosis. It is in the infected environment in which they live that the children of the tuberculous become contaminated. Rational prophylaxis will protect them from the disease.

A. Ghon and B. Roman add another contribution to the pathologic anatomy of tuberculosis in childhood, touching the matter of the propagation of the tuberculous process from the primary pulmonary lesion to other tuberculous processes in the body. They first quote a case of Ganghofer's and show how it confirms Ghon's previously reported theory of a primary pulmonary lesion and helps toward the solution of the new phase of the question. This case was one of acute tuberculous meningitis of the base of the brain, with a few acute miliary tubercles in the spleen, lungs, and liver and older tuberculous lesions in the lungs and their lymphatics. A small, date-like, caseous area in the right lung was evidently the primary lesion, as this lesion and those in the draining lymphatics were the most advanced and as there could be no other explanation of the acute miliary tuberculosis of brain, liver, and spleen than rupture into the blood stream.

Ghon and Roman report six cases, all confirming Ghon's early theory of a primary pulmonary lesion and all showing that extension

of the tuberculous process takes place through the lymphatics draining this area. In five of the cases the primary lesion was on the lateral part of the interlobular surface of the right upper lobe. The lymphatics of this area pass through the bronchopulmonary lymph nodes to the lymph nodes in angles of division of the bronchi, thence to the nodes on the anterior surface of the right pulmonary hilus. The difference in the changes in the interlobulary nodes between the upper and middle lobes and those between the middle and lower lobes proved this point, as did also the difference between the upper and lower tracheobronchial glands. Of not less significance was the difference in size of the anterior bronchopulmonary glands at the hilus and the posterior ones. The greatest changes were always in the glands situated in direct topologic relation to the involved pulmonary area.

As regards the course of the tuberculous process, the authors divide the six cases into two groups. In Group 1, including three cases, there was no tendency to heal. The tuberculous process was a progressive one. The fact that the tuberculous process can remain for a time limited to the portal of entry and its outgoing lymphatics, is important in considering tuberculosis in childhood.

Group 2 also included three cases and there was a tendency in some of these lesions to heal, but they all showed progressive lesions as well. The question of the relation between the progressive lesions and those with a tendency to heal is interesting. Was the progressive lesion an exogenous new infection or an endogenous recrudesence? Hamburger has pointed to the importance of this question from a clinical standpoint and from the anatomical one it is not easy to answer, nor is it easy to determine which lesion is primary, where two or three pulmonary lesions exist. It is doubtless true that two pulmonary lesions may be due to distinct exogenous pulmonary infections or one may be primary and the other secondary from aspiration from the primary lesion. The authors think that these questions can not now be definitely answered and that progress will be made only when exact anatomic examinations are made, with these questions kept clearly in mind.

A. Frisch[7] reports the necropsy findings which fulfill the theory of Ghon and Roman in regard to the spread of the disease, in a case of extensive tuberculosis in an 8 weeks-old baby. The oldest lesions were three cheesy pneumonic areas in the left lung, two of which were probably the result of a lymphogenous infection from the first. From these areas the disease had spread to the left tracheobronchial glands, then evidently to the right tracheobronchial glands, as caseation was less advanced in this than in the first group. In the mucous membrane of the left bronchus were tuberculous changes which were the

result of contact infection from the pulmonary lesion. In the middle ear were lesions of hematogenous origin, as were also in the mucous membrane of tonsil, adenoid tissue, tongue and epiglottis. These may, however, have been due to contact infection from sputum. In direct relation to the lesions in the nose and throat, were the tuberculous changes in the submental, submaxillary, retropharyngeal and cervical glands.

The tuberculous changes in the stomach and the small and large intestines were more recent than those elsewhere in the body, and consisted of miliary tubercles. They were either of hematogenous origin or due to contact infection from sputum which had been swallowed. Changes in the mesenteric glands were the direct result of the intestinal infection.

In the spleen, liver, pancreas and adrenals were miliary tubercles of hematogenous origin. Besides the three primary lesions in the lung, there were many miliary tubercles also of hematogenous origin.

F. H. Bartlett[8] analyzed 178 necropsies, in which tuberculosis was found. He concluded that in children the primary infection of the lung was the usual mode of infection, since 82 per cent. of the cases were of the pulmonary type. The absence of tuberculous lesions in the lungs in fourteen cases, and the presence of such lesions in the bronchial lymph nodes in seven of these, seemed to show that it was possible for the tubercle bacilli to pass through the lungs without localizing there. The largest number of cases were of inhalation origin, as shown by the large percentage of cases in which the pulmonary lesions were the most advanced in the body.

The marked tendency to rapid and general dissemination of tuberculous lesions in infants was well shown. In view of this fact the survival of any young infant infected with tuberculosis for more than six months after infection was very remote, while healing in young infants was practically out of the question. In the entire series, not a single healed tuberculous lesion was found, and attempts at healing, as shown by calcified areas, were observed in only five instances in the lungs and thirteen in the lymph nodes. Only twice was it found in infants less than a year old, and in both instances, the extensive generalization of the tubercles throughout the body gave evidence that the small calcareous areas present did not prevent the spread of the infection.

The comparative frequency of tuberculous meningitis in young infants was noteworthy as evidence of the very great general dissemination of tuberculous lesions in young subjects. There was a smaller proportion of cases of tuberculous meningitis among children who acquired the disease by ingestion than among those who acquired

it by inhalation, which finding was in keeping with the fact that the cases infected through ingestion showed rather less tendency to generalization than did the respiratory cases.

F. Hamburger[10] differentiates primary infection in tuberculosis from reinfection. Primary infection usually takes place from a sufferer of phthisis who has tubercle bacilli in the sputum. The person infected is usually a child. This is proved by the following facts: Children who live with tuberculous individuals almost without exception react to tuberculin and, when children less than 2 years old become ill with tuberculosis, they have associated with some one with tubercle bacilli in the sputum. The opinion that infection can be carried from cattle to man is not positively proved. It seems inconceivable that infection can be carried in this way and there are no reports in the literature of instances of direct infection of infants from cattle with tuberculosis of the udders.

Primary tuberculous infection usually takes place by inhalation. This statement is founded on the law of primary lesion and regional lymphatic gland involvement, as well as animal experimentation. Animal experiments show that infection with tubercle bacilli simultaneously causes lesions at the point of inoculation and in the lymphatic glands draining the infected area. Moreover, when the disease has become advanced, the changes in the glands are more widespread and severe than at the point of inoculation.

Primary infection takes place easily. Association for a short time with an individual who coughs is sufficient to produce infection. Hamburger quotes an instance of a child being in a room with a consumptive only half an hour and dying a few weeks later of tuberculosis. Primary tuberculous infections during the first year are very serious, but the seriousness decreases from year to year, so that primary infection after the sixth year is without danger.

Reinfection with tuberculosis is either endogenous or exogenous. Endogenous infection may be bronchogenous, hematogenous, or lymphogenous. Bronchogenous infection results from mucus being carried from the primary lesion to other portions of the lung during a spell of coughing. The mucous membranes of the larynx or nasopharnyx may also become infected in this way.

Hematogenous and lymphogenous infection results from rupture of the primary lesion into the blood or lymph vessels.

When tubercle bacilli penetrate the tissues of an individual already infected, there is a reaction between the specific antibodies and the tubercle bacilli (an allergeic reaction). If the reaction is energetic, all or most of the bacilli are killed, so that no lesion develops immediately, but after months or years of quiescence an exacerbation may occur. Often in this case lesions in the glands draining the area of

reinfection become active before the area of reinfection itself. Very frequently, however, no exacerbation ever occurs, something which shows that immunity to reinfection does exist.

Exogenous reinfection is almost without exception an inhalation infection. It occurs very frequently, especially in individuals who are associated with patients with phthisis. Many persons develop an immunity to exogenous reinfection, while for many it is very dangerous.

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The experience of E. Rumpf and J. Zeissler[11] has shown that there is a great difference in the results of finding tubercle bacilli in the blood by microscopic examination and by animal experimentation. In nearly every case examined they found bacilli microscopically, while only 8.5 per cent. of the animals inoculated developed tuberculosis. The conclusion is that the bacilli found microscopically in the blood were not virulent tubercle bacilli, especially as, in spite of all care, they could not grow on artificial mediums. It is held, however, that occasionally careful examination will reveal tubercle bacilli in the blood of tuberculous individuals.

A. Jousset[12] calls attention to the fact that when a guinea-pig is inoculated with tubercle bacilli, a bacillemia develops before a lesion. He suggests that a similar bacillemia occurs in the human, and, in fact, finding after death, the tubercle bacilli in the heart's blood of a number of infants, he has been able to prove that such a bacillemia does exist in infants. That the tubercle bacillus does not live a saprophytic existence excludes a cadaveric contamination with this organism.

Jousset's observations were made on six infants from 5 to 10 weeks old and on two from 6 to 7 months. Clinically they presented the ordinary picture of an athrepsia with gastro-intestinal disturbances. The cutaneous reaction was usually negative. Jousset says that in a breast-fed baby a regular decline in weight with a persisting diarrhea, which cannot be explained, suggests a tuberculous bacillemia, and he believes a large part of infant mortality attributable to this condition.

A. Philip Mitchell,[13] continuing his studies begun in 1911, investigated the relative frequency of the human and bovine type of tubercle bacillus in lymph glands, obtained at operations and at necropsies, on children.

The investigation consisted of gross examination of the glands, inoculation of guinea-pigs with emulsion of glands, and microscopic examination of glands.

The glands of twenty-nine children below 12 years were obtained at necropsy, but cultures of tubercle bacilli grew only in twelve instances; eight were the human type of bacillus and four the bovine. In the four instances of infection with the bovine type there was a more extended caseation of the mesenteric glands than of any other group.

The bronchial and mesenteric glands of eighteen children showing no evidence of tuberculosis at necropsy were inoculated into guinea-pigs. Tubercle bacilli were demonstrated in but a single instance in the bronchial glands of a child aged 4. The bacilli were of the human type.

The glands of eighty children were obtained at operation. Forty-two of the children resided in Edinburgh, thirty-eight in the neighboring country. In seventy-one, or 88 per cent., the bacillus isolated was of the bovine type; in nine, or 12 per cent., of the human type. Most of the children were under 2 years of age, and 84 per cent. had been fed unsterilized milk since birth. In the majority of instances, except for the glandular enlargement, the children were in robust health and gave no other clinical evidence of tuberculosis. The influence of the acute infectious diseases, as apparently favoring the dissemination of the disease in the glands, was noted in fifteen cases. In not a single case was there a history of pulmonary tuberculosis in other members of the family.

In conjunction with this study, Mitchell[14] investigated the prevalence of tubercle bacilli in the milk supply of Edinburgh. Of 406 samples of mixed milk, eighty-two, or 20 per cent., contained tubercle bacilli.

Mitchell draws the following deductions from his work: The results of inoculation with necropsy material, because the cases were few, cannot be considered conclusively as to the frequency of bovine and human types of infection, but they furnish evidence that in fatal tuberculosis in children the human bacillus is the main contributor to the mortality rate. Having demonstrated that a considerable proportion of the tuberculosis affecting children in Edinburgh is of bovine origin, and that the milk supply of this city is frequently infected with tubercle bacilli, and having demonstrated that a certain number of deaths occur from this bacillus, he no longer regards bovine bacilli as a negligible factor in the spread of tuberculosis among children.

Mitchell hopes that the high degree of tuberculosis contamination which he has found in the Edinburgh milk supply may be an effective argument for those who are working with the legislature in the cause of pure milk.

H. L. Coit[15] is of the opinion that it has not yet been determined to just what extent bovine tuberculosis is dangerous to human beings. The introduction of tuberculous infection into a herd of cows sometimes occurred when new stock were purchased. It seemed that dealers sometimes "plugged" their cattle with tuberclin before selling them, so that they did not react to the tuberculin test. To avoid danger from this source, it should be made compulsory to brand every animal that reacted to the test. It is also desirable that owners of dairy farms should breed their own cattle for replenishing their stock. The government spent many thousands of dollars each year making tuberculin tests for private individuals and yet refused to cooperate with the Milk Commission, entirely failing to appreciate that by cooperation far better results could be obtained at a much reduced expenditure.

Coit reviewed the investigations made in New York City, by Dr. Hess, and, in England, by Dr. Anderson, with reference to the incidence of bovine tuberculosis among children using ordinary uncertified milk. The results of their investigations seem to show that the danger of infection with the bovine bacillus was very slight, though all agreed it did occur. The writer urged members of the Milk Commission and other organizations interested in a pure milk supply, to take more interest in the work and recommended that, for the purpose of making the tuberculin tests, only veterinarians having the standing of a professor in a veterinary college be employed.

F. M. Pottenger[16] thinks that it has been fairly demonstrated to the satisfaction of the scientific world that: 1. Bovine infection can be transmitted to human beings. 2. While bovine infection is accountable for about 10 per cent. of tuberculosis, human infection is accountable for most cases in the human race. 3. While bovine infection is common, it is rarely found in adults.

In regard to this difference in frequency of bovine tuberculosis in children and adults, Pottenger says we must believe either that bovine infection for the most part becomes nonvirulent or heals, confining its sphere of activity to the child, or that the bovine bacillus changes its type by residence in the human body. What seems most probable to the writer is that the bovine bacilli, after living in human tissue for a time, adapt themselves to the new conditions, and gradually become the bacilli which would naturally be produced in human soil.

A. de Besche[17] reports an instance of infection with both the human and bovine type of tubercle bacilli. He says it is well known that there

has existed a marked difference of opinions as to how atypical strains of tubercle bacilli isolated from cases of tuberculosis are to be regarded. A number of investigators have declared that the strains revealing atypical characteristics are transitional forms between the human and bovine types.

It has been stated by the English Tuberculosis Commission in their final report that strains which appear as transitional forms cannot be so regarded, because a closer study of cultures may show that the bovine and human tubercle are mixed.

In the writer's case the culture was isolated from a mesenteric gland of an 8 months-old child, who had died of general tuberculosis. On isolation in the usual way, the bacilli showed the cultural peculiarities characteristic of tubercle bacilli of the human type. Testing the virulence, however, indicated a virulence for animals not corresponding to the human type. Further experiments proved the presence of both the human and the bovine type of tubercle bacilli.

DIAGNOSIS

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23. Salvetti, G.: The Significance of the Cutaneous Reaction in the Diagnosis of Tuberculosis in Children, Abstr. Jahrb. f. Kinderh., 1915, xxxii, 173.

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25. Rogers, O. F.: A Study of Children with Positive Skin Tuberculin Reactions, Boston Med. and Surg. Jour., 1915, clxxii, 161.

26. Fishberg, M.: The Cutaneous Tuberculin Test in Children of Non-tuberculous Parentage, Arch. Pediat., 1915, xxxii, 20.

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28. Young, W. A. B.: Tuberculous Infection in Young Children with Special Reference to Its Occurrence in Cases of Malnutrition, Jour. State Med., London, 1915, xxiii, 21.

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35. Lackner, E., and Levinson, A.: The Permanganate Test for Spinal Fluid, with Report of a Case of Tuberculous Meningitis where all other Tests were Negative, Arch. Pediat., 1915, xxxii, 508.

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According to T. Frazer,[18] our ideas as to the frequency and significance of the v. Pirquet tuberculin test have suffered change. Recent experiments seem to indicate that the test may be not only of diagnostic value, but also preventive.

A positive cutaneous reaction is less frequent in children than it was once thought to be. The former high percentage of positive reactions was due to the fact that the test was applied to infected children of the poorer classes. Also a difference in technic may play a part in the variation of the number of positive reactions. While there is an increasing percentage of reactions with years, the view held that the reaction has significance only during the first two years of life, is not borne out by recent figures, and all reactions occurring up to the age of 10 years should be regarded as suspicious.

With the recognition of this increasing infection and also of the relation which exists between infection in childhood and clinical tuberculosis in later years, it follows that, if the infection is detected and treated, a great stride forward will be made in the prevention of clinical tuberculosis. The applicability of the test, then, is to detect the presence of infection at the earliest possible moment, and to this end its employment annually would contribute in no small degree.

B. S. Veeder and M. R. Johnston[19] conjectured from their work at the St. Louis Children's Hospital that positive tuberculin reactions were much less frequent than was generally supposed. To discover whether this was a misapprehension or a fact they went over the clinical records of the past three years, tabulated the results of the tuberculin tests, and summarized as follows: A study of tuberculin tests in 1,321 hospital children in St. Louis shows that the percentage of positive reactions reaches a maximum of 44 per cent. between the years of 10 and 14. (Cases of clinical tuberculosis are included.) Where clinical tuberculosis was excluded, the percentage for this age period is only 36 per cent. These figures are much lower than the usual 90 per cent. figure, which has gained such widespread publicity and which is based on the figures of Hamburger of Vienna.

The extent of infection among children varies in different cities and countries and is dependent on such factors as living and social

conditions, the amount of tuberculosis in the community, and the exposure of the child to open tuberculosis. In all probability the extent of infection also varies among different classes of society in the same community. No conclusions as to the extent of infection with the tubercle bacilli can be drawn from statistics of one city or class and the statement that 90 per cent. of individuals are infected by puberty is an extreme exaggeration of the actual condition.

W. R. Ramsey[20] gives his results of v. Pirquet reactions made with old tuberculin and bovine tuberculin on children in the State Hospital for Crippled and Deformed Children in St. Paul. In all 116 children were examined, fifty-eight boys and fifty-eight girls. Whenever a positive reaction was present for one tuberculin, it was present for the other also.

Of the fifty-eight boys, twenty reacted positively and thirty-eight negatively. Of the fifty-eight girls, twenty-two reacted positively and thirty-six negatively—a total of forty-two positive and seventy-four negative reactions.

Seventeen boys and eleven girls gave a negative v. Pirquet, when the clinical diagnosis was tuberculosis of the bone. Of the total number of fifty-eight diagnosed clinically as tuberculosis, only twenty-eight gave positive v. Pirquet reactions.

M. S. Voronin[21] tested 140 infants for the v. Pirquet tuberculin reaction, with undiluted tuberculin and with dilutions of 5, 10, 15, 25, 50, and 75 per cent. One hundred and thirty of the infants were between 2 days and 4 months of age; ten were between 6 months and 1 year. Of the 140 tested there were only two positive reactions and those were but weakly positive, one with undiluted tuberculin and one with a solution of 75 per cent.

M. N. Keifetz[22] examined 276 children of various ages for the v. Pirquet reaction to tuberculin and found it positive in 53 per cent. The children who lived in favorable surroundings gave a positive reaction only half as often as children living in poor and unhygienic surroundings. The v. Pirquet test is specific for tuberculosis and indicates a tuberculous infection. Keifetz thinks it desirable to examine all school children by this simple and decisive method.

G. Salvetti[23] vaccinated 630 children by the v. Pirquet method; 166 gave a positive, 327 a negative and 37 a doubtful result. Children who showed clinical evidence of tuberculosis gave negative reactions in 18 and doubtful reactions in 8 per cent. of the cases. Of twenty-one cases of tuberculous meningitis, fifteen gave positive reactions, also two cases of erythema nodosum. If the children contracted measles, the previous positive reaction became negative in a large per cent. of the cases.

J. B. Manning and H. J. Knott[24] performed the v. Pirquet test on 228 children, 166 of whom had been exposed to tuberculosis and sixty-two of whom had not been exposed.

Eighty-four of the 166 children, who had been or were at the time of examination still living in intimate contact with adults suffering from active tuberculosis, gave an active tuberculin test. Sixty-four, or 82 per cent. of these showed no clinical evidence of the disease; fifteen, or 17.8 per cent., had lesions in varying degrees of activity. There were seven children with bronchial gland tuberculosis, one with active keratitis, two with cervical adenitis, four with distinct lung invasion, and one with tuberculous peritonitis.

Eighty-two of the group of children who had been exposed to tuberculosis gave a negative v. Pirquet reaction.

Of the sixty-two children who had not been exposed, fourteen, or 22.5 per cent., gave positive reactions and forty-eight, or 77.4 per cent., gave negative reactions.

In the entire series of 228 cases with so many known exposures it is interesting to note that the percentage of reactions was only 42.9 per cent. These figures do not bear out Fishberg's assertion that there is but slight difference in the number of positive reactions among those who live in a tuberculous mileau and those who do not.

O. F. Rogers[25] inquired into the history of sixty-nine children subsequent to their discharge from the Massachusetts General Hospital, where they had given a positive v. Pirquet reaction. The history inquired into covered a period not less than one year, and not more than four years. He was able to ascertain the fate of fifty of the sixty-nine. Seventeen of these, of whom eleven, or 65 per cent., had died, were below 2 years of age; four, or 23 per cent., showed no evidence of active tuberculosis; and only three were really well. Thirty-three were over 2 years of age, and of these only seven, or 21 per cent., had died. The outlook was, then, nearly three times as favorable in the second as in the first group.

Rogers draws the following conclusions from his study: A positive cutaneous tuberculin reaction before the age of 2 seems to indicate that the child's life is likely to be short. The mortality among all children under the age of 10, who react to the v. Pirquet test, is much higher than among those who do not.

M. Fishberg[26] applied the tuberculin test to 588 children under 15 years of age, in whose homes there was no person known to have tuberculosis. His aim was to ascertain whether only children raised in a tuberculous environment run the risk of infection with tuberculosis, while children living under similar social, economic, hygienic and sanitary conditions, but whose parents are nontuberculous, escape infection.

Of the 588 children 310, or 52.7 per cent., gave positive reactions. The test was applied only once and the author is of the opinion that, if it had been applied a second time to those who did not react the percentage of positive reactions would have been increased 10 to 20 per cent.

Fishberg says there are several problems to be discussed in connection with these findings. It is of immense interest to know whether a positive reaction is an indication of infection with the tubercle bacillus; if so, how have these children become infected; and finally, what is the outlook for these children—are they destined to suffer from clinical tuberculosis sooner or later? All available evidence seems to show that these infections during childhood are harmless to the vast majority of persons. The fact that so many give positive reactions to tuberculin and yet are healthy is sufficient proof that infection with the tubercle bacillus is not alone enough to produce phthisis. Moreover, Fishberg thinks that these mild lesions are not only innocuous, but even beneficial, since they protect the individual from exogenous reinfection. Persons who have not undergone a mild infection during childhood, when infected with the tubercle bacillus, are likely to develop hematogenous tuberculosis of a rapidly fatal type.

Fishberg makes the following summary of his article: The cutaneous test satisfactorily shows whether a person has been infected with the tubercle bacillus or not, but it is of little value in the diagnosis of the disease known as tuberculosis in its various manifestations. In the large cities and even in the small rural communities most of the children have been infected with tuberculosis before reaching adolescence, though they have not necessarily suffered as a result of this infection.

G. H. Cattermole[27] performed the cutaneous tuberculin test on a number of children in Colorado, in order to ascertain, if possible, the frequency of this disease among children in that state.

Of sixty-six children tested, twenty-five, or 38 per cent., gave positive reactions; forty-one, or 62 per cent., gave negative reactions. Thirty-four of these children had tuberculous parents, while twenty-nine had parents free from tuberculosis. In spite of the fact that 50 per cent. of the children had parents who had suffered from active tuberculosis, only 38 per cent. of the children showed a positive reaction at the time of examination.

In twenty-two instances tuberculous children had tuberculous parents. In twenty-seven instances nontuberculous children had nontuberculous parents. In twelve instances nontuberculous children had tuberculous parents. In one instance a tuberculous child had nontuberculous parents.

Of the twelve children who did not react, but who had tuberculous parents, three were less than 2 years old; one was 2 years old; one

was 4 years; one 5 years; three were 6 years; one was 7 and two were 9 years old.

Cattermole draws the following conclusions from his study: Children who are infected with large numbers of tubercle bacilli, as those living in the tenements of the cities, or near active pulmonary cases, develop hypersensitiveness, as shown by the tuberculin reaction.

When children have never been infected, or in cases in which the number of bacilli received is very small and the child's resistance good, as in Colorado, they do not react and are either not infected or are immune.

Possibly all tuberculosis is contracted in childhood and from three-sevenths to six-sevenths of the population acquire immunity at this time; while the others succumb to the more acute tuberculous conditions of childhood or later show the chronic pulmonary type of adults, which is believed never to occur except in those who have been infected and rendered partially immune in childhood.

In the prevention of tuberculosis the aim should be to protect infants from all exposure, and older children from massive infection. Moderate and gradual infection in older children probably insures immunity.

W. A. B. Young[28] in the Manchester Children's Hospital has tried to ascertain the incidence of tuberculosis among infants suffering from malnutrition and not giving any clinical evidence of the disease. He performed the tuberculin test on 113 of them. He found a positive reaction in thirty-one, a negative in seventy-seven, and a doubtful in five. No child under 3 months gave a positive reaction, and only one under 6 months. Of fifty-five children under 1 year, seven gave positive reactions; of eighty-five under 1½ years, fifteen gave positive reactions; of ninety-three children under 2 years nineteen gave positive reactions. Thus, a considerable number of marantic children, with no clinical evidence of tuberculosis, gave a positive tuberculin reaction.

G. T. Palmer[29] discusses the possibility of a tuberculin reaction in the child after the drug has been administered to the mother, and cites a case in point. A woman suspected of having tuberculosis was given 1 milligram of tuberculin intradermally. The next day her six months-old, nursing baby was reported to have developed bronchitis or bronchopneumonia and a week later definite meningeal symptoms. Examination showed extensive pulmonary involvement and meningitis. The child died ten days after the test had been given to the mother.

Considering the likelihood that the child was tuberculous, as he had been cared for frequently by a father who had open pulmonary tuberculosis, and considering the infrequenty of any effect on the child of the administration of tuberculin to the mother, Palmer thinks it highly probable that the relation of the test to the illness of the child

was purely one of coincidence. This view is supported by the fact that for two or three weeks before the acute illness the child had been indisposed.

The literature on the effect on the nursing child of a tuberculin test given the mother is very meager, but seems to confirm the theory of coincidence. Though there is no question of the fact that toxins as well as immune substances may be transmitted through the milk, it seems hardly probable that in this case sufficient tuberculin could have reached the child to cause the slightest disturbance.

The author finds the case interesting and suggests that possibly greater care should be exercised in giving tuberculin to the nursing mother, and that the element of coincidence should be fully considered when unfortunate complications follow its use.

J. A. Colliver[30] experimented to ascertain whether the skin in one part of the body is more susceptible to tuberculin than another. He tested fifty children and selected the dorsum of the hand and foot, the inside of the knee, the shoulder, the neck, the back, and the ulnar side of the forearm for the site of vaccination. Undiluted tuberculin was inoculated with the v. Pirquet drill. Measurements and observations were made every two hours after the inoculation for the first fourteen hours, then, beginning the next morning, every four hours for the next twelve hours, and then every six hours, until the reaction reached its maximum.

The conclusion drawn from the experiments was that there is no good reason for making the v. Pirquet tubercular cutaneous test elsewhere than on the forearm, and that this location has the advantage of being convenient.

D. B. Lees[31] reports four cases of incipient tuberculosis in children and emphasizes the value of percussion properly performed, as an aid in diagnosis. He says, however, that, to arrive at a sound conclusion, it is essential to adopt a correct method. The inadequate and misleading statements of textbooks in regard to percussion in incipient tuberculosis are founded on examination of patients in sitting or standing posture. This posture invariably leads to false conclusions in the examination of the front of the chest, because it does not allow of complete relaxation of the muscles or of the counter resistance, which is necessary for even the lightest percussion stroke. It is essential for the examination of the front of the chest that the patient assume a recumbent position on a comfortable couch. For the examination of the back of the chest he should be in a sitting and slightly stooping posture, with his hands on the front of the opposite shoulders, the elbows pressed together, and the muscles relaxed. The physician should press firmly with the terminal phalanx of his left hand on the

spot to be examined, keeping all the rest of his hand and arm away from the patient's chest wall. He should then percuss lightly with the finger of his right hand and measure in finger breadths the diameter of the dull area.

In any case of phthisis it is possible to detect the existence of a typical series of dull areas, which are found in the same position in case after case. In an early stage of the disease, when the symptoms are suggestive but not conclusive, the discovery of the typical series of dull areas is sufficient to establish a diagnosis. Simple collapse of the lung, common in young children, is irregular in distribution and not arranged in the pattern characteristic of tuberculosis. Bronchopneumonia of nontuberculous origin affects mainly the lower parts of the lungs. In the early stage of pulmonary tuberculosis the entry of air is very defective over the typical dull areas and there may be no morbid sounds. This fact is likely to cause error in diagnosis. Even at a late stage, when the areas are large, there may be a few or no morbid sounds.

W. Overend[32] describes the shadows seen in Roentgen-ray plates of children with tuberculosis. A diffuse shadow extending from the vertebral ends of the third rib to the sixth rib is due to enlargement of the tracheobronchial glands and may be called tracheobronchial or superior opacity. A shadow just below the level of the sixth rib, running directly into the hilum and obliterating the narrow isthmus between the hilum and the cardiac opacity, is caused by congestion of the bifurcation glands and is called the intermediate opacity.

Shadows in the interspaces are due to perivascular and peribronchial tuberculous infiltration. Sometimes these interspaces become more translucent during inspiration and the shadows in the interspaces seem to suggest a close interlacement of lymphatics or a fine pleuritic exudate.

Shadows at the hilum are due to swollen, caseous, or calcified glands and the density of the shadows increases in this order. If the hilum is broad, irregular in outline and uniformly diffuse or cloudy in appearance, the process is active.

In the pulmonary field may be seen a fine shadowy network or shadows of small tubercles or large tuberculous foci. A general mottling is a sign of phthisis but not of activity, as it may be due to a healed tuberculous lymphangitis. Morton and Owen think that mottling is due to the crossed shadows of thickened lymphatic vessels. The occurrence of small shadows the size of a pinhead, standing separately or in conglomerate groups, producing a cloud effect, is diagnostic of activity. If fibrosis takes place, the opacities are more dense and sharply defined.

J. L. Morse[33] discusses the subject or what constitutes tuberculosis in childhood. He emphasizes the importance of distinguishing between tuberculous infection and tuberculous disease. For all practical purposes the infant comes into the world free from tuberculosis. In round numbers 10 per cent. of all infants become infected with tuberculosis before the end of the first year of life, and at 16 years, not more than 10 per cent. have escaped infection. However, a very small percentage of these children have tuberculous disease. Those who are well and show no evidence of symptoms of tuberculous disease are little, if any, worse off than those who have not been infected. In fact, it is probable that they are better off in that they have established an immunity to tuberculosis.

A positive tuberculin test signifies in itself nothing more than tuberculous infection; it does not denote tuberculous disease. Tuberculous infection in childhood is almost invariably glandular. In the struggle to overcome the infection the glands become enlarged and indurated. When the cervical glands enlarge, they can be seen and felt. The mesentery glands can be felt if sufficiently enlarged, while the tracheobronchial glands, although neither visible nor palpable, give rise to certain definite physical signs. d'Espine's sign which, when present, is said to indicate tuberculous tracheobronchial glands, shows that there is some enlargement of these glands, but shows nothing as to the nature of the enlargement. If a positive d'Espine sign is associated with a positive v. Pirquet reaction, the chances that the enlargement is tuberculous are increased. These chances are further increased if the sign is persistent.

The significance of Roentgenographs which show a shadow in the region of the tracheobronchial glands is exactly the same as that of a positive d'Espine's sign. Great care should be taken in interpreting Roentgen-ray plates. Only the opinion of an expert is of much value, and even that is not infrequently wrong.

There is little room for difference of opinion as to the presence or absence of tuberculous disease of the cervical glands or of the bones. The greatest opportunity for opinion as to whether a child has or has not tuberculous disease, arises when a child showing a positive tuberculin reaction manifests symptoms of general constitutional disturbance, such as fever, rapid pulse, loss of weight and strength, debility, fatigue, anemia and dry cough, without any evident signs of disease. The presence of a definite history of exposure to tuberculosis is strong evidence in favor of an active tuberculous focus somewhere.

Too much attention must not be attached to a slight elevation of temperature, for this is a very common thing from minor causes. In the writer's opinion, it is much more likely to be due to infection in

the nasopharnyx or the accessory sinuses, pyelitis or indigestion, than to tuberculosis.

Disturbances of nutrition, anemia, debility, fatigue and malaise are all symptoms of indigestion, overwork in school, late hours, lack of fresh air and many other things, as well as to tuberculosis. A dry cough may be due to nasopharyngeal irritation, enlargement of the lingual tonsil and various other local irritations, as well as to tuberculous tracheobronchial adenitis.

It is evident, then, that the presence of these symptoms does not justify a diagnosis of tuberculosis in the absence of physical signs of disease, even if they are associated with a positive v. Pirquet reaction and the diagnosis of tuberculosis should only be made after a careful and unprejudiced investigation and consideration of all the factors in the individual case.

J. B. Hawes[34] gives a number of points to be borne in mind when the diagnosis of tuberculosis in children is being considered. He says it must be remembered that there is a difference between tuberculous infection and tuberculous disease. Eighty to 90 per cent. of individuals are infected with tuberculosis before the age of 15 years, but the fact that the great majority live at least an average lifetime shows that this does not necessarily imply disease; that tuberculosis in childhood is primarily a disease of the glandular system, while in infancy it takes the form of a general septicemia; that bovine as well as human sources are of importance in considering exposure; that in childhood, by the time the lungs are involved, the disease is far advanced; that constitutional signs and symptoms are of importance. The principal ones are ease of tire, loss of weight or failure to gain weight, pallor, malnutrition and debility; that, while in adults there is no characteristic cough, in children there is a cough, which is more or less characteristic of bronchial gland enlargement; that a positive tuberculin reaction is of infinitely greater importance in children than in adults; that, though the Roentgen-ray in children may show enlarged bronchial glands it does not prove these glands to be necessarily tuberculous.

Hawes says also that the physician must not fail to realize the responsibility of stamping a child tuberculous, and that, if in doubt, he would be wise to institute proper treatment, without casting the stigma of tuberculosis on the child. Also it must be borne in mind that diseased tonsils, and increased adenoid tissue, a chronic middle ear inflammation, and other conditions may simulate tuberculosis.

E. Lackner and A. Levinson[35] describe the permanganate test for spinal fluid and report its value in determining the diagnosis of a case of tuberculous meningitis. The test depends on the oxidizing power of the permanganate, and is performed by titrating the spinal fluid

with a decinormal potassium permanganate solution. The spinal fluid in normal individuals or in those suffering from meningismus has little organic substance and the permanganate index is consequently low. In patients suffering from meningitis the organic substances are increased and the permanganate index is higher.

The author's case was that of a boy 7½ years old. The history, clinical findings, and especially the course of the disease were suggestive of tuberculous meningitis, but all laboratory findings (with the exception of one doubtful Noguchi late in the disease) were negative, except the permanganate test, which showed an index of 3.4 at the first examination. This indicated a tuberculous meningitis and necropsy confirmed the diagnosis.

H. Sundt[36] quotes Calot to the effect that it would be better if physicians in charge of sanitariums for the tuberculous would think oftener of syphilis, and if syphilologists would think oftener of tuberculosis. Sundt is chief of the seashore sanitarium at Fredricksvern, and he says that in a comparatively large number of cases the patients sent to the hospital are not suffering from tuberculosis. In forty cases of hip joint disease admitted during the last three years, 27.5 per cent. proved to be nontuberculous, as also 20.7 per cent. of twenty-nine cases of diseases of the knee joint. In nearly all of the nontuberculous hip joint cases the trouble proved to be Perthers' juvenile osteochronditis deformans, and four out of the six of the knee joints affections were also syphilitic. Since the beginning of 1915, five of the twenty-two new admissions to the sanitarium responded positively to the Wassermann test. They had been sent in with the diagnosis of tuberculous disease of the hip joint, foot, knee or larynx.

The patients in the hospital were mostly children, and latent syphilis masqueraded as tuberculosis or "scrofula" in a surprising large number of cases, or accompanied cases of unmistakable tuberculosis. Sundt gives a description of several cases illustrating this point. One is as follows: A child, who had both knees affected after a trauma, had long been treated for tuberculosis and a destructive lesion in the palate had been treated for lupus. The tuberculin reaction was positive as was also the Wassermann reaction. Antisyphilitic treatment soon restored the child to health.

Other cases cited show that syphilis alone can induce the classical picture of "scrofula" in its typical form. All these patients presented merely the late manifestations of inherited syphilis. In none of them had it been recognized, but tedious treatment had been enforced for tuberculosis or lupus.

There are no absolutely reliable differentiating clinical signs; a primary localization of tuberculosis in the nasopharynx is of very rare

occurrence. The tuberculous lesions generally spread superficially, the syphilitic burrow deep. The tuberculin reaction is most instructive, when it induces a focal reaction, but this is rather an exceptional occurrance. When the findings are dubious, a course of antispecific treatment may decide the diagnosis, but it must be remembered that not everything which subsides under potassium iodid is syphilis. Moreover, the Wassermann reaction may be doubtful even with known syphilis.

E. H. Risley[37] has attempted by an analysis of a series of necropsy and clinical records to throw some additional light on the preoperative diagnosis of tuberculous mesenteric and retroperitonael glands. The type of this disease which is secondary to general or intestinal tuberculosis is not considered, but only that type which has come to be recognized as a separate clinical entity.

Risley gives the following description of the disease, but his study did not enable him to add much to its preoperative diagnosis: The disease is extremely frequent in children, but by no means confined to this period, being nearly as common in young adults. As regards symptomatology, there may be no symptoms even in the presence of palpable glands. The condition may be discovered only at operation for some other condition, the glands being too small for palpation through the abdominal wall. Or there may be trivial symptoms, as fleeting but not severe right sided or general abdominal pain, with or without disturbance of digestion and bowel regulation. The general health is good or the patient is of the pale, "sickly" type. At times there is more or less constant, fairly well localized abdominal pain, with a soreness in the region of the cecum. Examination may or may not show the presence of resistance or an ill-defined mass. Or there may be sudden or gradual onset of, first, general, then localized abdominal pain, confined to the right lower quadrant. With the pain comes a rise of temperature, nausea, vomiting, constipation or diarrhea. Examination shows localized tenderness with spasm and resistance. The leukocyte count is between 12,000 and 15,000.

It is evident, then, that tabes mesenterica, like appendicitis, the lesion with which it is most often confused, gives a variety of symptoms and signs, none of them distinctive in themselves and from the analysis of which a preoperative diagnosis cannot be made.

SYMPTOMATOLOGY

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K. Fraser[38] has analyzed 296 cases of pulmonary tuberculosis among school children and makes important deductions from the results.

His statistics seemed to point to some relation between commencement of school life and the incidence of tuberculosis, and to the fact that school life has a prejudicial effect on children predisposed to tuberculosis. Children able to resist the attack so long as they are running about in the open air, break down when forced to spend a part of each day in stuffy overcrowded classrooms.

An inadequate increase rather than a failure to gain in weight is important. Closely associated with the question of weight is that of nutrition. The manifestations of impaired nutrition are many—pallor, anemia, weariness, loss of appetite. It is highly probable that defective nutrition is the first visible sign of tuberculous infection.

Night sweats in children may have no significance, and only, when they are described as cold and clammy, does the author attach any importance to them.

Cough as a diagnostic symptom is somewhat empirical, a persistent hard, dry cough is the type most to be suspected.

In regard to the localization of the lesion, one or both apices are usually involved. When only one apex is affected, it is more frequently the right, and, when both are affected, the right shows a more extensive lesion than the left.

Dulness is the most important physical sign. Inequality of breath sounds is an early indication of trouble, but the duration of this sign is short. Bronchial breathing is nearly always present and remains for an indefinite period. Friction and fine crepitant râles are the most significant accompaniments, and all accompaniments are suspicious, when peristent and localized.

Spontaneous improvement is more common in girls than in boys; from the sixth year the tendency to spontaneous improvement increases year by year. Children in rural districts show a very much greater tendency to spontaneous improvement than children in the cities.

J. P. Cullen[39] compares pulmonary tuberculosis in childhood with phthisis of adult life and finds the path of infection is identical. In both, the terminal bronchioles are the portal of entry. In the adult, possibly because of a blocking up of the lymph paths by a previous tuberculosis or mechanical irritation, the bacilli remain localized and produce a bronchiolitis or bronchopneumonia, which undergoes the usual changes of caseation, fibrosis, calcification, or softening. In childhood the infection does not remain localized; the lymphatics are involved, and a definite infection occurs along their entire course.

Signs and symptoms depend on the point of arrest of the bacilli. In the adult type this is at the beginning of the journey and local symptoms, like cough, expectoration, hemoptysis, are those for which the patient usually seeks advice. Physical signs, due to the situation of the lesion, are sufficiently well marked to facilitate an early diagnosis by the usual methods.

In childhood local symptoms are indefinite. There may be reflex cough and vomiting. General symptoms are more marked. The patient is languid and anemic and may suffer from night sweats or slight evening pyrexia. Growth and nutrition are interfered with. Physical signs, because of the depth of the lesion, are by no means obvious.

Enlargement of the normal interspinous, dulness with prominent venules over the region from the first to the fifth dorsal spines, crepitations in the nipple region, and harsh cogwheel sounds at the apices are the sounds which suggest further examination—as by means of the Roentgen-ray.

Adult and infantile phthisis are therefore one and the same disease. The difference in their symptoms and signs are explicable on anatomic peculiarities. The affection in childhood may remain limited to the lymphatics and then the prognosis is good, provided the disease is recognized early and treated properly. Diagnosed wrongly or too late, the parenchyma of the lungs becomes involved and the case passes on to the adult variety with slight chance of recovery.

According to O. W. McMichael,[40] our knowledge of early manifestations of tuberculosis in childhood must come from the formation in our minds of a composite picture, resulting from systematic study of large numbers of children who have been in contact with open cases of tuberculosis. This opportunity is offered in our Municipal Tuberculosis Dispensaries, where it is the custom to examine systematically all members of the family where one member is found to be tuberculous.

Inasmuch as the first resting place of the tubercle bacillus is always in lymphoid tissue, it is to the glands that we must give our first attention in the examination of children for tuberculosis. Glandular enlargement is found in nearly all children who are in contact with open cases of tuberculosis, while this is universally true of children so exposed, it is also true that these glands are enlarged in nearly all children. The presence of enlarged glands by no means prove tuberculosis. Hypertrophy of glands is evidence of combat against infection, and, if there is any distinction between the glands of tuberculous children and the enlargement of glands due to other infections, it is that in tuberculosis the palpable glands are smaller and firmer than those of nontuberculous children, and may be traced along the course of lymphatics as small, firm beads. These glands tend to disappear as the evidence of pulmonary involvement increases, so that, in children with extensive pulmonary involvement, the glandular enlargement may be very slight and distinctly less than the enlargement in the average normal child. The author has come to regard the disappearance of glandular enlargement without the development of pulmonary signs as evidence that the condition was nontuberculous, and, in tuberculous children, the shrinking of the glands with an increase in their density as evidence of favorable progress toward control of the disease.

The tendency to regard anemia and malnutrition as evidence of tuberculosis is very likely to lead to error, for, in family groups of children known to be exposed to open cases of tuberculosis, it is exceedingly common to find that the best nourished and least anemic

looking children are the ones who present the most signs of tuberculous disease.

One of the first things noticed by the parents is that the child stops growing. The tuberculous child is usually undersized and underweight. The posture in standing is relaxed and the alae scapulae are prominent in nearly every instance. The hair and skin are dry in children who show pronounced signs of the disease. No matter how well nourished, the tuberculous child has a weary expression about the eyes, the lower lid may droop sufficiently to show a line of sclera below the cornea, and McMichael has long noted a bluish-pink triangle with the lower lid as its base, which he regards as a distinct sign of tuberculosis in children.

The pupils of the tuberculous child are usually dilated. If the pupils are unequal one may suspect pulmonary activity on the side on which the dilation is more marked.

In examination of the chest, attention is first drawn to dilation of the superficial veins, usually denoting marked enlargement of the mediastinal glands. A further evidence of this enlargement is Smith's sign, which consists of a systolic murmur heard at the right border of the manubrium when the head is thrown back. The chest examination differs little from that of the adult, with the exception that it reveals the greater frequency of tuberculous deposits in the bases of the lungs in children than in adults. The more general the bronchitis, the less likely is this an evidence of tuberculous disease, while fine moist râles confined to a small area in the lung may be regarded with grave suspicion.

McMichael thinks that no matter how expert one may be in the interpretation of physical signs, more direct evidence of tuberculous disease can be obtained by the use of tuberculin than by any other means. The v. Pirquet skin reaction has the same significance in adults as it has in children. The mere statement that a v. Pirquet reaction is positive or negative gives little information, but the character of the reaction gives valuable information regarding defensive processes going on in the blood of the patient. A poorly nourished child under 1 year of age in the arms of a tuberculous mother will usually give a negative v. Pirquet reaction. A tuberculous patient who gives a slight reaction will, after the continued administration of some preparation derived from the tubercle bacillus, give reactions of increasing intensity which coincide with other physical signs of improvement, and, on the other hand, the tuberculous patient who is losing ground will show reactions of decreasing intensity and will finally fail to react at all. A negative reaction in a child is not conclusive evidence that the child is nontuberculous any more than a positive reaction is evidence that the child is the victim of tuberculous disease. The value of the reaction is dependent on the skill of the observer.

The use of tuberculin in the treatment of children promises more than does any other remedial measure. Children bear large doses of tubercle protein better than adults, and, to stimulate the production of immunity, appreciable doses must be given. This is especially true where operative procedures are contemplated and where the blood of the child must be capable of taking care of any tubercle bacilli that may be squeezed out into the circulation by the manipulation incident to the operation. It is unwise to undertake any surgical procedure until a child can endure a dose of 0.4 or 0.5 milligram of tubercle protein without severe reaction.

While working at the Woolwich Tuberculosis Dispensary, J. M. Campbell[43] placed tuberculous children in four groups. In the first, or suspect group, were all who, on repeated examination, showed physical signs in the chest, such as poor expansion at one or the other apex or of one lung generally, with impaired resonance over the same area, and weakened air entry. In this group were also placed children who showed the stigmata of tuberculosis and who were living in homes where there was an adult with open tuberculosis.

In the second group were placed those in the incipient stage, the most difficult to diagnose. In many instances there were no more physical signs than in the cases of the first group, but there was evidence of toxemia. The child was pale and had lost weight; there was sometimes a short cough and sweating at night. The temperature curve varied more than normal. Among the physical signs on which the author laid stress were granular or "sticky" inspirations, the presence over the suspected area of fine râles, which did not disappear on coughing, and a positive v. Pirquet test.

The third group includes cases of caseous and fibrocaseous tuberculosis. The signs and symptoms in these were very similar to those found in adults—but the lesions were less often apical and frequently more extensive at the root of the lung or at the base. The toxemia in this form was less marked in children than in adults and in the fibrocaseous form contraction of the lung caused little inconvenience to the child.

In the fourth group were placed cases of tuberculosis of the bronchial glands and of hilus tuberculosis. These formed a fairly large group and gave definite signs and symptoms. There was usually an indefinite and progressive condition of ill health in a child who had been healthy. There was anemia and lassitude; the muscles were flabby, and the child suffered from a troublesome paroxysmal cough. Physical signs were found only when there was pressure on a bronchus which prevented inflation of the lung.

The writer decries the tendency definitely to label too many children tuberculous. She thinks it necessary to revise and standardize

the nomenclature of tuberculosis, and that until this is done there will be much indiscriminate notification of cases to the detriment of the child.

B. S. Pollak[44] calls attention again to the similarity of tuberculosis and syphilis. He says that in tuberculosis as well as in syhilis there is a primary stage with primary symptoms and regional lymphatic gland involvement, a secondary stage which runs through a course of years with tendency to relapses, and a tertiary or late stage which is known as phthisis pulmonalis. Also, tuberculosis, like syphilis, causes immunity against reinfection.

H. Koch[48] defines the early stage of infection with tuberculosis as the period between the day of infection and the time when the v. Pirquet reaction becomes positive. By a systematic application of the v. Pirquet test Koch was able to determine this early stage in three patients, and by careful study of their symptoms he attempted to find clinical signs which would offer a possibility of diagnosis of this stage.

His three cases were as follows: *Case 1.*—An infant was admitted to the hospital three hours after birth. The mother had advanced tuberculosis. In the eighth week at the hospital the child developed a temperature and in the ninth the cutaneous test became positive. *Case 2.*—An infant three weeks old was admitted to the hospital. The v. Pirquet test was negative. In the seventh week there was a rise in temperature and the v. Pirquet test became positive. *Case 3.*—A four and a half year old boy was brought to the hospital because of a non-tuberculous inflammation of the hip joint. He ran a temperature for two weeks. The v. Pirquet test was negative when the boy was admitted, was repeated systematically, and became positive after three months.

The three patients had only one symptom in common—a rise in temperature at the time the v. Pirquet test became positive. The type of temperature corresponded to that found in the last stages of the disease, namely, an evening rise with a morning remission. The author calls this febrile reaction the initial fever of tuberculosis. In spite of careful observation, no other symptoms were found, and there were no local signs, due to the pulmonary lesion or involvement of the regional bronchial lymphatic glands. But in Case 3 the Roentgen-ray pictures taken after the infection, showed a more marked and widespread shadow at the left hilus than the first one taken before the infection.

It is evident that systematic tuberculin tests and measurement of temperature can not be made on healthy children, but to make them on children who remain a long time in hospitals and are exposed to tuberculous infection would be of theoretical interest and of value. These children should be watched for the onset of the infection and for

clinical symptoms during its early stages. In the writer's cases the initial fever and the positive v. Pirquet test occurred in the seventh week after the infection.

H. Bergmann[49] reports a case of advanced pulmonary tuberculosis with cavity formation in a 2-months-old child whose mother had died of tuberculosis shortly after his birth. The child was brought to the hospital March 2, 1914, with a history of having coughed since birth. He had never been nursed. Examination showed a poorly nourished baby, with a temperature running between 36.6 and 39.2 C. Over the left lower lobe posteriorly were large moist râles. The v. Pirquet test was positive. The child died March 22, 1914. Necropsy showed the following: Both lungs were adherent to the thorax at the apices; over the visceral pleura were many miliary tubercles. In the upper part of the right lung was a large walnut-sized cavity with pus-like contents, besides many small pea-sized cavities. Throughout the entire lung were small and large partly caseated tubercles. In the lower part of the upper lobe of the left lung were two cavities, and scattered throughout this lung also were many caseated tubercles. The bronchial glands were enlarged and caseated. One kidney and also the cecum contained many miliary tubercles.

In discussing the case, Bergmann first considers the positive tuberculin test in so young an infant. He quotes Friedberger and Simmel, who found in newly born sensibilized guinea-pigs a pronounced hyposensitiveness to a reinjection, and Rietchel, who thinks that antibodies can be carried from the mother to the fetus, but exist only a short time, and Schanz, who also has shown that antibodies can be carried from the mother to the offspring in guinea-pigs, but are soon lost. Bergmann, then, believes that the child of a mother who gives a positive tuberculine test, will also give a positive reaction soon after birth, but for a few days only. If the child is tuberculous, the positive reaction will reappear.

Of the pathologic findings, Bergmann notes especially that the lungs were widely diseased and the other organs hardly affected at all. He thinks the case one of congenital tuberculosis hematogenous in orgin, and explains the extraordinarily severe manifestations by the active functionating of the lungs at birth.

F. Hall[50] reports a case of rapidly advancing pulmonary tuberculosis in a 2-year-old child, whose mother was known to have the disease since its birth. The child had been breast fed by the mother for thirteen months. When 6 months old she contracted pneumonia, complicated by otitis media, but apparently she made a complete recovery and remained well until two weeks before entrance to the hospital, during which time she had been quite ill. There had been a discharge from the eyes,

nose and ears; she had had elevated temperature, had coughed considerably and had lost weight.

The diagnosis on entrance as far as the lesion in the lungs was concerned, was a diffuse bronchitis, but the Roentgen ray made evident the presence of a tuberculosis, and as the disease progressed, showed its extension from the hilus into the lobes. This was more marked in the right lower lobe. The Roentgen-ray picture showed also the presence of a cavity at the left hilus before it was evidenced by physical signs. The cavity formation was confirmed at necropsy.

The v. Pirquet test had given a marked reaction early in the disease. It became faint as the child became weaker, and absent a few days before death. Tubercle bacilli were not found until late in the disease, when the diagnosis was well established. They were found first in the stools, then in the sputum.

H. Barbier[51] reports a case of acute tuberculous alveolitis in a 3-months-old infant, who was moribund when brought to the hospital. At necropsy the lungs were congested and had the appearance of being filled with miliary tubercles. There was also a caseous bronchial adenopathy.

Histologic examination showed an intense alveolitis, intra-alveolar exudation and infiltration of the thickened alveolar walls. The exudate was composed of amorphous material resulting from necrosis of the alveolar epithelial cells. In some places there was complete destruction of the alveolar walls. Sections stained by the Ziehl method showed an enormous number of tubercle bacilli within the alveolae, and in the necrotic tissue of alveolae. Barbier believes that in this case the infection took place by inhalation.

F. Weihe[52] has made an interesting clinical and roentgenologic study of interlobular pleuritic exudates in children. He does not agree with Eisler, who attributes these exudates always to a tuberculosis, but believes they are often metapneumonic in origin. Of a series of eight cases, observed within two years, there was only one which he regarded as tuberculous. This patient was a 3-year-old, emaciated boy who had a tuberculous involvement of the larynx as well as marked signs in the lungs. The Roentgen-ray picture showed a widening of median shadow and irregular shadows scattered through the right pulmonary field. On the right side, running from the right rib upward to the fifth, was also a sharply defined linear shadow, which evidently represented an old interlobular exudate.

In all the other instances the children were acutely ill. There was a high evening temperature, with a morning remission. This febrile period lasted from eight days to three weeks and usually ended abruptly. The few pulmonary findings contrasted sharply with the

severe general condition. Sometimes a band of dulness was found on percussion in the axillary region. In the author's cases the exudate was usually on the right side and the Roentgen-ray picture showed a horizontal shadow, the thickness of a lead pencil, beginning at the lower border of the fifth rib in front and ending at the lower border of the fourth rib laterally. In some instances after the shadow had run about one-third of its course, it spread out fan-like and formed a triangular shadow on the chest wall. Repeated pictures showed that these shadows absorbed rapidly.

Whenever the exudate could be outlined by percussion or by the Roentgen ray, an attempt at puncture was made. In four cases this was successful and small amounts of cloudy pus were obtained.

Weihe then divides interlobular exudates into two groups, the metapneumonic and the tuberculous, and he is not sure that it is always possible to differentiate between the two. He thinks, however, one point in differentiation is that tuberculous exudates absorb with difficulty, while metapneumonic exudates absorb quickly and their shadows are seen to disappear in a series of Roentgen-ray plates.

E. Germonig[53] reports a case of tuberculous pharyngitis in a 22-months-old infant. There was no case of tuberculosis in the child's direct family, but one sister of the mother had died of the disease. Examination showed a fairly well developed infant. The muscles were flabby and there were enlarged glands on both sides of the neck. The lungs were resonant throughout and the breath sounds were sharp and rough, especially over the lower lobes posteriorly. The Roentgen-ray picture showed a dark area at the hilus of the right lung, which evidently corresponded to a group of bronchial glands. In the throat there was ulceration of the uvula, hard palate, and palatoglossal arches. The epiglottis was much thickened. Examination of the larynx was impossible, but from the clear cry of the child, it was evident the vocal cords were intact. The v. Priquet test was positive. Microscopic examination of a small piece of tissue excised from the uvula gave a picture typical of tuberculosis. No tubercle bacilli were found.

D. S. Wilson[54] reports the case of laryngopharyngeal stenosis, complicating pulmonary tuberculosis in a 16-year-old boy. The unusual feature of the disease was that the base of the tongue, the epiglottis, the pharynx, and the larynx were incorporated in a mass of adhesions. The aperture through the pharynx to the esophagus was reduced to about the size of an ordinary lead pencil. It had become impossible for the boy to swallow, and liquids, instead of reaching the esophagus, regurgitated through the mouth and nose. Respiration was seriously impaired by the stenosis and the whistling sound produced by the boy's breathing could be heard for a considerable distance.

A. E. Meyers[55] analyzed 105 cases of tuberculous meningitis, because from textbook descriptions it is difficult to estimate the frequency of the various signs and symptoms.

Of the 105 patients, fifty-three were females, fifty-two males. Over 30 per cent. were between 2 and 3 years of age, and over 80 per cent. between 18 months and 5 years. Most of the cases occurred in June, July and January. Measles was mentioned in the past history of twenty-seven patients or 26 per cent.; whooping cough in 22 per cent. Thirty-eight per cent. had had no previous infectious disease.

The average duration of the disease was seventeen days. The average period in the hospital five and eight-tenths days. This varied directly with the number of lumbar punctures. Tubercle bacilli were found in the spinal fluid in 21.5 per cent. of the cases. The fluid was under pressure in 80 per cent. The average cell count was 198 cells to the cubic millimeter. The prevailing type of cell was the small mononuclear ranging from 90 to 100 per cent. in 67 per cent. of the cases. In three cases the polymorphonuclear leukocyte was the prevailing type of cell. A fibrin clot was present in 70 per cent. of the cases. A positive globulin test was obtained in 50 per cent. of the cases; in 7 per cent. it was negative; and the test was not mentioned in 43 per cent.

The lowest white blood cell count was 8,500, the highest 48,000. The largest percentage of cases showed a count between 10,000 and 15,000.

Of the cases in which a v. Pirquet test was performed 63 per cent. gave a positive reaction, 25 per cent. a negative and 2 per cent. a suggestive reaction.

There was a definite history of direct exposure to tuberculosis in 25 per cent.; no known exposure in 73 per cent. and no record of exposure in 2 per cent.

In 30 per cent. of the cases there were sufficient signs in the lungs to show they were involved in the tuberculous process. In two cases d'Espine's sign was noted.

In 71 per cent. of the cases the eye reflexes were abnormal. In over 30 per cent. the patella reflexes were equal and active; in 21 per cent. they were absent; in 5 per cent. they were equal and sluggish; in 6 per cent. they were irregular; in 6 per cent. they were not mentioned; in 32 per cent. they were equal and normal. There was a positive Babinski sign in 21 per cent.; a negative in 55 per cent. The condition was not mentioned in 24 per cent. Abdominal and epigastric reflexes were positive in 14 per cent., negative in 17 per cent. and not mentioned in 69 per cent. Brudzinski's sign was positive in 40 per cent. Kernig's sign was present in 27 per cent. Forty-three per cent.

of all cases showed some kind of paralysis. Rigidity of the neck was present in 70 per cent. of the cases.

Thirty-nine of the patients had convulsions before entrance; forty-five were unconscious on entrance. In practically every history drowsiness, apathy, mental dulness, languor, irritability and sleepiness were mentioned. In over 85 per cent. vomiting was recorded as an initial symptom. Pain was a comparatively frequent symptom. Headache was present in 17 per cent. In 50 per cent. there was a history of constipation. Grinding the teeth, restless sleep, delirium, loss of sphincter control, crying, capricious appetite and inability to swallow were symptoms frequently mentioned.

In 41 per cent. of the cases there was a final rise in pulse, temperature and respiration curves. A definite remission of symptoms with a corresponding drop in pulse and temperature occurred in 64 per cent. There were no recoveries.

C. O. Hawthorne[57] discusses localizing brain symptoms in tuberculous meningitis and reports two cases. He says that occasionally tuberculous meningitis has an abrupt onset, and that this may take the form either of localized palsies or localized convulsions. His first case was that of a boy 10 years old who had at one time had some enlarged glands, but otherwise a negative history. On September 18 the child was said to have been sick while at school, but on the two following days was as active as usual. During the night of the twentieth he again vomited and in the morning examination showed a paralysis of the left face and left upper limb, which disappeared within the course of forty-eight hours. On the following day there were repeated convulsive seizures, limited to the left limbs, and inability to speak. On the cessation of the convulsions, left hemiplegia was again observed. Later the paralysis entirely disappeared, but headache, persistent vomiting and temperature remained and these with the finding of abundant cellular elements in the cerebrospinal fluid left no room for uncertainty in the diagnosis of tuberculous meningitis.

The second patient was a boy 1 year and 7 months old. He was suddenly seized with convulsions, which were strictly limited to the right side. These ceased in an hour or so. There was no paralysis or evidence of organic disease and the cerebrospinal fluid was free from excess of cells and micro-organisms. Two days later the convulsions recurred and the child gradually developed characteristic signs of tuberculous meningitis. The diagnosis was confirmed by necropsy.

Hawthorne offers as an explanation of the unusual onset in these cases that from some primary source as the mediastinal glands, an embolus or multiple emboli reached the brain through the blood stream. It is not difficult to see how this sudden blow might be followed by a local recovery of nerve function, with consequent disappearance of the

convulsions and paralysis. The subsequent appearance of the signs of a generalized meningitis must be explained by a tendency of the specific irritant to produce a gradually extending infection.

H. Brockmann[58] reports two cases of pseudomeningitis in tuberculous children, offering great difficulty of diagnosis. The first was that of a 2-year-old boy, with a family history of tuberculosis. The child had always been well until three months before his admission to the clinic, Feb. 28, 1914. For the previous week, he had been restless, slept badly, cried a great deal and had frequently put his hand to his head.

Examination showed a relatively well nourished child. Scattered over the body were many small papular tuberculids. There was an infiltration of the left apex with weakened respiration and moist râles. The v. Pirquet test was at first doubtful, later weakly positive. There were also symptoms which pointed to involvement of the central nervous system. Marked hyperesthesia, sensitiveness to light, tense and bulging fontanel, right convergent strabismus, obliteration of right nasolabial fold, pronounced rigidly of the neck, paresis of right upper and lower extremities, hypertonia in all, especially in both right extremities, patellar reflex increased on the left side, diminished on the right, patella clonus increased on both sides.

Lumbar puncture March 2, gave a slightly cloudy fluid, under pressure, albumin content six lines, according to the Niszl method. In the sediment were many lymphocytes but no tubercle bacilli could be demonstrated. On the basis of these clinical findings and of the characteristics of the spinal fluid, the writer made a diagnosis of tuberculous meningitis and at the same time suspected the existence of a tubercle, localized in the left hemisphere, and explaining the right sided hemiplegia which had existed three months.

The child's condition remained stationary for months. For several weeks 50 c.c. of fluid were withdrawn every second day, always showing the same inflammatory changes. Twice it was injected into guinea-pigs, but the animals lived. This unusual course caused the writer to abandon the diagnosis of tuberculous meningitis and make one of solitary tubercle situated in the left ventricle, which, by inflammatory irritation caused a rich secretion of lymphocytes and albumin. The child died September 21, and necropsy confirmed the diagnosis. In the neighborhood of the left cerebral ganglion was a conglomerate tubercle pointing toward the enlarged cerebral ventricle. There was also a marked internal hydrocephalous and a fresh crop of miliary tubercles on the meninges, but no evidence of a healed meningitis.

The second patient was a 9-weeks-old child, also with a family history of tuberculosis, admitted to the clinic Oct. 23, 1914. The baby was well nourished; scattered over the body were many small papular

tuberculids. The upper and middle lobes of the right lung showed well marked infiltration. The v. Pirquet reaction was positive. The fontanel was tense and there was pronounced hypertonia of the lower extremities, with increased patella reflexes. Lumbar puncture yielded a clear fluid under high pressure, but with a normal albumin content and no increase in lymphocytes. No tubercle bacilli were found and animal experiments were negative. Necropsy showed wide-spread tuberculous changes in the lungs and caseation of the bronchial and mesenteric glands. The brain and inner membranes were normal; the dura showed patches of pachymeningitis.

Both these cases gave a clinical picture of tuberculous meningitis. Iu the first it was produced by a giant tubercle in the neighborhood of the cerebral ventricle, which had given rise to localized inflammation of the ventricular wall. In the second it was caused by a meningismus, due to the tuberculotoxic products.

The author urges that caution be exercised in making a diagnosis and giving the prognosis in conditions which simulate tuberculous meningitis, for complete cures can be effected in such as he cites.

E. B. Friedenwald and W. Greenfeld[59] report an unusual case of tuberculoma of the brain in a 7-year-old colored boy. The child was first seen in June, 1912, when he had had a convulsion. Other convulsions, epileptiform in character, always beginning with spasm of the right arm and leg, followed at irregular intervals. During the summer the convulsions became more and more frequent, the child's appetite failed, and he lost weight rapidly. Toward the end of autumn the convulsions became very violent. On Jan. 11, 1913, after a violent convulsion, the patient became acutely ill, the temperature rose, the respirations became rapid. Patches of crepitation were found over the whole right lung. On the twenty-first the abdomen became rigid and contained fluid. The child died Jan. 24, 1913.

Necropsy showed caseous pneumonia of the right lung, fibroid apex of left lung, tuberculous peritonitis, and tuberculous bronchial glands, and small tuberculous foci in the liver. The most interesting changes were in the brain, in both hemispheres of which a number of dense, whitish, grayish masses were embedded. The left cerebral hemisphere contained a number of conglomerate foci forming a large mass, situated in the ascending parietal and superior marginal and superior parietal convolutions. The right cerebral hemisphere contained four masses, one in the upper part of the mid frontal convolution, one in the lower part of the external surface of the occipital lobe, the third in the parieto-occipital sulcus, and the fourth, extending across the limiting sulcus, involved the upper part of the insula, the claustrum and external capsule. Histologically the masses showed the ordinary changes produced in tuberculosis, but most unusual were the masses of tubercle

bacilli found in the vessel walls and in the caliber of the vessels of the brain and bronchial lymph glands. (The authors could discover in the literature no other instance of tubercle bacilli found in the blood vessels.)

The original focus the authors think was the fibroid apex of the left lung or the tuberculous bronchial lymph glands, from which the infection was carried to the brain. The twitching of the right arm and leg was explained by the tumor in the left ascending parietal convolution.

L. Guthrie[60] reports a case of tuberculoma of the pons varolii in a 3½-year-old boy. The child was admitted to the hospital Feb. 2, 1915, a year after an attack of pneumonia. He was gradually losing weight, his cheeks were frequently flushed, and a cough, which had been present on and off since birth, had become worse. Strabismus had developed in the left eye ten days before admission.

Examination on admission showed a thin, feeble child with a dusky flush on his cheeks. He was dull and apathetic but intelligent. The chin constantly deviated toward the left side and the head toward the right shoulder. The optic disks and fundi were normal, but the left eye was strongly turned inward. No outward movement beyond the median line was possible. Power of inward movement of the right eye was also lessened. There was complete paralysis of the left side of the face. Apparently only the left sixth and seventh cranial nerves were paralyzed. There was no spasticity of the lower extremities, but the knee jerks were rather more active on the right than on the left side. The left apex was dull on percussion, the breath sound over this area was almost amphoric in character and accompanied by loud, coarse râles. Many râles were heard over both bases. The v. Pirquet test was markedly positive.

The child lived six weeks after admission and showed slight change of condition. At necropsy the pons was found to be occupied in its center by a tuberculous mass about 1 inch in diameter. As the brain was removed from the base of the skull, a small, soft, caseating tumor about the size of a cherry was left attached to the cribriform plate of the ethmoid. Two or three similar masses were found just beneath the cortex of the frontal lobes. Throughout the lungs were many miliary tubercles. The bronchial glands were distinctly tuberculous.

De Stefano[61] reports a case of tuberculous cardiocirrhosis in a child of 10 years. For two years she had had febrile symptoms and headache, followed by abdominal swelling. For one year she had had strabismus and convulsive attacks repeated at long intervals. On admission to the hospital examination showed cyanosis, an enlarged and hard abdomen with a palpable spleen. The precordial region was prominent and increased in size. The bases of both lungs were dull, with weak

vesicular murmur and absence of vocal fremitus. There was also dulness in the right supraspinous fossa with weak inspiration. There were drowsiness, increased patella reflexes, and a positive cutaneous reaction. The Wassermann reaction was negative. At necropsy there was found a polyserositis, universal pericardial adhesions, nutmeg liver and tuberculous masses in the brain. The case may be considered one of polyserositis, causing pericardial adhesions and nutmeg liver.

H. Barbier[62] describes a form of tuberculosis of the liver in children suffering from hereditary tuberculosis, analogous to hereditary syphilis of the liver. The liver in these cases shows extra- and intralobular perivascular sclerotic changes accompanied by fatty degeneration of the liver cells. The chief characteristic, however, is the hypoplasia. In some cases the degree of hypoplasia of the liver corresponds to the degree of atrophy in the child; in others it exceeds it. In almost every case miliary tubercles or confluent tuberculous nodules are found associated with the above changes.

Barbier reports a case of this form of hepatic tuberculosis. The patient was a 3½-months-old infant whose father had tuberculosis. The child had seemed well for a month and a half, when it had ceased to gain in weight. For two months before entering the hospital he had suffered from cough, vomiting and fever. He was in the hospital ten weeks and during that time there was a diffuse bronchitis, anorexia, and emaciation. At necropsy there was found a caseous bronchopneumonia and a generalized miliary tuberculosis. But what was most striking was the aspect of the liver. It was small (165 gm.), yellow and literally riddled with tubercles, the size of millet seeds. Microscopically there were many caseated tubercles and a number of the hepatic lobules were completely caseated. There was a diffuse hepatitis with cellular necrosis, but the liver cells did not show fatty degeneration. Very remarkable was the great number of tubercle bacilli in the center of the nodules.

E. Feer[63] has studied the small papular tuberculid which occurs in children and he is surprised at its frequency. The eruption consists of slightly raised papules, usually 1 or 2, rarely 3 or 4 millimeters in diameter, which do not itch. In the center of the papule is a brown scale or depression. The waxy glistening character of the wall around the central scale or depression is quite characteristic and is noted especially by reflected light or when the skin is put on a stretch. Suppuration of the center of the papule makes a differential diagnosis from acne difficult. The lesion may last weeks and then disappear completely, or leave a flat scar.

Papular tuberculids are usually few in number and occur on the thigh, in the groin, in the gluteal region, and on the extensor side of

the arm. They are found more often in younger than in older children —and more frequently in infants. They are the result of embolism, either of tubercle bacilli or of toxic products. In one instance Feer found tubercle bacilli in histologic sections and produced typical tuberculids by inoculation of the material into guinea-pigs.

The finding of papular tuberculids usually signifies that an active tuberculosis is present.

TUBERCULOSIS OF THE TRACHEOBRONCHIAL GLANDS

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E. Rist[64] asks: 1. What trace is found in the adult of glandular tuberculosis of childhood? 2. How account for the difference between the lesions of the bronchial glands of the child and those of the adult?

He says that the relation between the pulmonary and glandular lesion in the child and adult are reversed. In the adult, extensive ulcerative tuberculosis of the lungs is generally found postmortem, coincident with bronchial glands of normal size, and vice versa in the child. Instead of being fixed to the surrounding tissues by strong fibro-adhesive bands, the glands are easily enucleated, and, what is more important, they are rarely affected by caseous degeneration as are those of the child. Moreover, most of the glands in the adult contain small hyalin masses often anthracotic and partly calcified. Rist thinks it is incorrect to assume that these glands have become sclerotic and are unable to react to any infection, for they do react to any disease except tuberculosis. Nor does he think that caseation of the bronchial glands is a specific characteristic of tuberculosis of the bronchial glands of childhood, but of a primary tuberculous infection. If pulmonary tuberculosis in the adult is not accompanied by caseation of the corresponding glands, it is because the pulmonary

tuberculosis in the adult is a reinfection. Caseation is not specific of tuberculosis of childhood, but of primary tuberculous infection.

W. W. Howell[65] has attempted to ascertain the frequency of chronic inflammation of the bronchial glands in infancy and childhood, also the reliability of signs of enlarged glands, and their possible pathology. With these objects in mind he examined for enlarged bronchial glands all the children who came under his care during one year. There were over 300 babies under 2 years admitted to the Infants' Hospital, 500 schoolchildren between 6 and 13 years, and patients in private practice.

The means used to determine the enlarged glands was auscultation, i. e., the determination of D'Espines sign, and percussion. The Roentgen rays were used to confirm the physical findings.

From his examination Howell accepted as a positive D'Espine a change in character of whispered voice sounds (in older children) or of the expirium during crying spells (in infants) at or below the third dorsal vertebra. He found percussion of the glands difficult, but dulness in the second interspace to the right of the sternum and dulness to the right of the vertebral column at the level of the spine of the scapula was definite and significant and agreed pretty well with the intensity of D'Espine's sign. The roentgenograms of patients with positive physical findings always showed shadows.

The author says there is a general belief that chronic bronchial adenitis means tuberculosis, and, while this may be true in infancy, this study has led him to believe it otherwise in older children. He thinks that in older children, if the enlargement of the glands is accompanied by anemia and malnutrition, the underlying condition is probably tuberculosis, but, as he frequently found enlarged bronchial glands in well nourished children with carious teeth, enlarged tonsils and adenoids and a chain of palpable crevical glands, he thinks these conditions are factors in the production of the enlarged bronchial glands.

T. Frazier[66] reviews the main diagnostic points on which the recognition of tuberculous bronchial glands depends. He thinks the history and symptoms are as important as the physical signs, if not more so. The history of exposure to infection or of an attack of measles, whooping cough, or influenza, with the nature of convalescence, is to be considered.

In the beginning of the disease the symptoms may be very indefinite. Loss in weight, or a stationary weight, is the most significant constitutional symptom. Pallor of the face and mucous membranes may appear so gradually that it is not noted until it has existed some time. There is a loss in strength and the child shows evidence of malnutrition and becomes languid and fretful. A cough is usually present, although there is no particular type of cough characteristic of tuber-

culous bronchial glands. In younger children it may be spasmodic, resembling whooping cough; in older children it is likely to be dry and unproductive.

The significance of enlarged cervical glands is not so great as it was once thought to be. Lymphoid activity is great during childhood and palpable lymph glands are the rule.

The v. Pirquet skin reaction has a decided value, especially in younger children.

The examination of the chest in children is often unsatisfactory and a diagnosis of tuberculous bronchial glands is arrived at with great difficulty. In the early stages there may be no physical signs, although the disease is active. In older children there may often be seen, when a deep breath is taken, the so-called hilus dimple, a depression anteriorly in the inner part of the upper two interspaces. The most constant sign on percussion is a slight dulness in the interscapular region. D'Espine's sign is of no importance unless it is heard below the level of the first or second dorsal spine in a child of 8, or below the third or fourth in a child of 12. Fine râles about the nipple are found in a number of instances.

Although none of the physical signs are absolute in their significance, the presence of dilated veins, interscapular dulness, whispered pectoriloquy beyond the normal limits gives the suspicion of tuberculous bronchial glands. The suspicion is strengthened if the child exhibits any or all of the symptoms mentioned.

E. Rach[67] adds another contribution to those he has already written on the diagnosis of tuberculous bronchial glands in children.

Rach agrees with other writers that in children, the glands in the right tracheobronchial space are the ones most frequently affected in this condition. Since his last report he has seen a great number of Roentgen-ray pictures of tuberculosis of these glands and has been able to verify the pictures anatomically. These pictures exhibit a uniformity worthy of note. The shadows representing the enlarged glands runs parallel with that of the trachea, and, above and below, joins the median shadow. The shadow is usually date or club shaped.

However, Rach does not believe that a diagnosis based only on radiologic findings is satisfactory, and he discusses a group of symptoms, due to pressure of the glands on neighboring structures, which, when taken with the Roentgen-ray pictures, is very significant.

This group of symptoms has frequently been described in the literature. Rach quotes Barthez and Rilliet, who mention rough paroxysmal cough, asthmatic attacks, hoarseness, without laryngeal involvement, from pressure on the nerves; intermittent cyanosis and edema of the face from pressure on the superior vena cava; and expiratory stridor from pressure on the trachea and bronchi.

The expiratory stridor seems to be one of the most important symptoms. It has been described by Schick as follows: It is heard during the entire expirium, more easily when the child is at rest. The expirium is lengthened and dyspneic — the inspirium barely audible. After a while the expiratory difficulty causes an emphysema of the lungs. On necropsy Schick found that the stridor was caused by compression of a principal bronchus by cheesy, enlarged lymph glands. Its expiratory character is due to the fact that during inspiration the bronchial tubes dilate and allow the air to enter, while during expiration the bronchial tubes collapse and increase, by the pressure of the enlarged glands, the obstruction already present. The reason for the occurrence of the expiratory stridor in young children only is that, after the first year, the bronchial tubes lose their elasticity and do not dilate and collapse so readily.

Rach, after the study of forty-one cases of enlarged bronchial glands, draws the following conclusions: Enlargement of the right tracheobronchial glands gives, in Roentgen-ray plates, a date- or club-shaped shadow, which bends outward from the median shadow and runs parallel with the trachea. In consequence of their topologic relations, these glands can cause distinct clinical symptoms by compression of the neighboring structures. In infants, compression of the right principal bronchus causes a crowing expiratory stridor, in older children a pertussis-like cough. These symptoms in conjunction with a positive cutaneous reaction and the characteristic radiologic findings, are sufficient to make a diagnosis of tuberculous bronchial glands.

H. F. Stoll[68] has made a study of D'Espine's sign of enlarged bronchial glands. In 1907 D'Espine drew attention to a sign that he had observed for many years and considered diagnostic of enlargement of the bronchial glands. He stated that in young children the whispered voice normally ceases at the level of the seventh cervical spine posteriorly. When, however, there is enlargement of the lymph nodes and the patient whispers "thirty-three," bronchophony is heard over the upper thoracic spine as well.

Stoll has had a limited opportunity to compare this sign with the postmortem findings. In a few patients not presenting any increased whisper sounds no enlargement of the bronchial glands was found. Four patients exhibited a marked D'Espine and all had large bronchial glands.

One case was of especial interest. The patient was a 10-year-old child sent to the writer because of enlarged cervical glands, which proved to be tuberculous. From physical signs a diagnosis of enlarged bronchial glands, involvement of the right upper lobe, and thickened pleura or effusion at the right base was made. The Roentgen-ray picture taken a few weeks later showed extensive disease at the right

root. All the time the child was under observation, whispered bronchophony was present in the interscapular space and was always most marked to the left of the thoracic vertebrae. The child died six months after coming under observation and at necropsy this area was found to be directly over an enlarged gland between the left lobes.

Stoll summarizes his views regarding D'Espine's sign as follows: Whispered bronchophony in the interscapular space is indicative of a pathologic process at the hilus of the lung. This may be due to enlarged glands, the result of malignancy, leukemia, Hodgkin's disease, syphilis, or any infectious disease of the lungs. Its presence in the delicate child is exceedingly suspicious of tuberculous involvement of the tracheobronchial glands.

Occasionally enlargement of the bronchial glands is present when there is no change in the whispered voice. The diagnosis of clinical tuberculosis, however, rests on the sum total of our physical signs and symptoms, not on one isolated sign.

S. Maggiore,[69] on examining thirty-seven children who gave a positive tuberculin reaction, found an area of dulness in the supraspinous and infraspinous fossae, in twenty-two instances on the right side, and in fifteen on the left. In twenty-eight, there existed more or less marked evidence of tracheobronchial adenopathy. Radioscopy showed in only four instances any shadow in the upper pulmonary lobes, and in these four cases moist sounds were heard on auscultation. In the remainder, radioscopy gave evidence of enlarged peribronchial glands on the same side as the dulness. There was a necropsy in four cases. In one, in which moist sounds had been present, the lung was healthy, but there were enlarged caseous glands in the region corresponding to the dulness. The conclusion arrived at by the author is that apical dulness in children who react to tuberculin is due, in the majority of cases, to tracheobronchial adenopathy, even when moist râles are present.

T. Gött[70] describes two cases in which he found an unusual shadow in the roentgenologic picture. The shadow was very similar to the one ascribed by Rach to abscess following a tuberculous spondylitis of the lower cervical or upper thoracic vertebrae. Rach's shadow was bow shaped and extended laterally, in the thoracic region, beyond the shadow of the vertebral column.

Gött's first patient was a 5-year-old girl who had developed a widespread pulmonary tuberculosis following a pneumonia and empyema. Examination on the child's entrance to the hospital showed besides the pulmonary findings, a rigidity of both legs, with increased patella and achilles reflexes, which suggested to the writer incipient meningitis. The Roentgen-ray picture, however, showed shadows which were evi-

dently those of a pulmonary tuberculosis, as well as a spindle shaped shadow, beginning somewhat higher than the bifurcation of the trachea and reaching to the diaphragm. It extended only slightly to the right of the spinal column, but decidedly to the left, so, although it did not conform strictly to Rach's shadow, a diagnosis of spondylitis of the lower cervical or upper dorsal vertebrae was made — especially in view of the rigidity of the lower extremities and the increased reflexes. With this diagnosis in mind the formation of a gibbus and a psoas abscess was predicted, but the diagnosis had to be changed, as six months later there was no evidence of either condition and the rigidity of the legs had disappeared. The Roentgen-ray picture still showed the abnormal shadow and in consideration of the necropsy in the second case, which proved that such a shadow could be caused by enlarged or broken down glands in the posterior mediastinum, the diagnosis of abscess from broken down tuberculous glands was made.

The author's second case was that of a 2-year-old boy, who entered the hospital with symptoms characteristic of a leukemia. The Roentgen-ray picture showed a thick shadow which began at the sternoclavicular joint and ran parallel to the vertebral column. Its median portions merged with the shadow of the vertebral column; its lateral borders were sharply defined and extended beyond the shadow of the vertebral column. Necropsy revealed a leukemic infiltration in the posterior mediastinum near the vertebral column, extending to the level of the tenth thoracic vertebra, which had evidently caused the shadow in the Roentgen-ray picture.

The localization of the pathologic conditions explains the similarity of the Roentgen-ray pictures of two very different processes, and also the similarity to Rach's shadow of spondylitic abscess, which differed from these only in contour.

Pesch[71] reports an instance of rupture of a tracheobronchial gland into the trachea. The patient, a 6-year-old child, was brought into the clinic because of difficulty in breathing. He had been treated about a year for tuberculosis of the knee joint, and he had been coughing a few days. On examining the child there were noted a marked dyspnea, cyanosis and a stridor. During the examination the dyspnea became so severe that tracheotomy was performed immediately, but the child received no relief. Then a forceps was introduced into the trachea through the wound and a small cheesy mass removed from the lower part of the trachea. The breathing became free immediately. The next day, however, the dyspnea and cyanosis returned and were again relieved by removal of another piece of glandular tissue. On the third day several more pieces were coughed up. The child died on the third day.

Necropsy showed miliary tuberculosis of all the organs, caseation of the bronchial and tracheal glands, one of which had perforated the lower portion of the trachea.

The author points out that whenever the diagnosis of perforation seems probable, tracheotomy should be performed and an attempt made to remove the caseated gland through the tracheotomy wound.

A. Wersen[72] has made some interesting observations — roentgenologic and clinical — on the treatment with calcium salts of bronchial gland tuberculosis. He says that in childhood the bronchial glands are the starting point of most of the tuberculous processes. The bronchial glands enlarge, the lymphoid tissues become necrotic, the capsule thickened and adherent to the surrounding tissues. If the capsule remains intact the tuberculous process heals (usually by calcification) otherwise it spreads rapidly into the lungs, or breaks into the blood vessels. A rational therapy must have, then, for its object a rapid cure of the process, while the capsule of the glands is still intact. For a long time it has been believed that in tuberculosis there is a marked loss of calcium and that this demineralization predisposes to the disease. It has also been thought that calcification is due to the formation of organic substances with increased affinity for calcium. The caseous necrotic masses in tuberculosis of the bronchial glands seem to have this affinity. The question is, can an increased calcium intake influence this affinity and hasten the calcification of the glands? The author has attempted to solve this problem by administering calcium to children suffering from tuberculous bronchial glands and by noting the possible changes, in a series of Roentgen-ray pictures.

Twenty children from 5 to 14 years of age were treated with calcium, nine with calcium lactate, eleven with calcium lactate and sodium tartrate, and the effects on the glands carefully noted in Roentgen-ray pictures. For comparison twenty children not treated with calcium were also observed.

In the first group calcification of the bronchial nodes was noted in eight or perhaps twelve of the patients, in the second series in one, perhaps two, patients. In the first series it was also evident that the hilus shadow became more distinct and more sharply defined from the neighboring tissues, in twelve, perhaps fourteen patients, and in only two of the patients in the second series. Also the clinical symptoms of the patients given calcium were favorably influenced.

On the whole the author is well impressed with the results of this treatment.

PROPHYLAXIS

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A. F. Hess[74] says that in the field of the mode of transmitting tuberculosis the clearer conception as to the time of life when infection takes place — early childhood — is the greatest achievement of the last few years. He raises the question whether this knowledge is profited by in establishing prophylactic measures against the disease. Making investigation in 120 families in which some member suffered from tuberculosis, he found among them forty-two infants under 2 years of age. These children are doomed to contract the disease and to disseminate it. Unless infection in young children is adequately guarded against, there is little hope of eradicating tuberculosis in succeeding generations.

As a remedy, Hess suggests the establishment of preventoriums for infants. To make a beginning in this direction, it may be advisable to assign wards for this purpose in existing institutions (infant asylums, preventoriums or sanitariums) rather than to establish new institutions. It would then be feasible to have in many instances infant and parent in the same institution. These infants need not be sent to the country to undergo a rigorous fresh air regime; they can be adequately cared for in or near the city, where their parents may visit them from time to time. Preference might be given at the outset to urgent cases, to infants surrounded by open tuberculosis, to cases in which a tuberculous mother is constrained to remain at home in order to care for her infant, or in which there are two or more members of the family suffering from tuberculosis. The minimum length of stay may provisionally be fixed at one or two years.

The object, however, of Hess' paper is not to formulate details of such a plan but to urge those engaged in tuberculosis and infant welfare work to make some provision for the large number of infants, who are now abandoned to their fate, destined to die, or to swell the ranks of the tuberculous poor in the coming generation.

Mary E. Lapham[75] gives some practical suggestions as to the prevention of tuberculosis. She says it is every day becoming more evident that by the time our children reach maturity they are all infected with tuberculosis, that therefore the attempt to protect our people against tuberculosis should be directed not so much against preventing an infection already and inevitably acquired, as toward protection from the consequence of what has already occurred and can no longer be averted. The terrible death rate from tuberculosis is not due to the presence of infection alone, but to the impossibility of knowing when the infection becomes dangerous.

Lapham quotes Allport, who says that there are twenty million schoolchildren in the United States and that one million have tuberculosis. In the present state of our knowledge, then, she says, it is useless to protect our children against this wholesale infection, because we do not know when or how it is acquired. All children are infected and 1 or 2 per cent. will die. The prevention of tuberculosis consists in saving the 1 or 2 per cent., by detecting the infection at the very start, and this can best be accomplished through regular periodic examination of children and adults by competent authorities.

New York City recently advised the yearly examination of each child in the public schools. If this were done by competent authorities, Lapham thinks we might learn that a surprising number of children suffering from tuberculosis regain their lost immunity through the help of open air schools and good food. Also that after a while we may learn that overcoming tuberculosis in the child will stop tuberculosis in the adult.

Lapham mentions the pitiable inadequacy of maintaining sanatoriums at public expense. The number of cases of manifest tuberculosis has not been reduced one whit by sanatorium methods, and can never be reduced by the expenditure of any amount of money, so long as we wait until tuberculosis manifests itself. The yearly examination of every child in the public school, if not ruined by political appointments, will detect when and where tuberculous processes start in the children, and eventually teach us that the danger from tuberculosis is far more from within than from without.

J. H. Peck[76] emphasizes M. E. Lapham's point that prophylaxis of tuberculosis can best be accomplished by routine examination of schoolchildren for evidence of early signs and symptoms of the disease. In

this way alone can the child be protected from the consequences of the infection.

In an editorial[77] in the *Archives of Pediatrics* is described the home hospital experiment of the New York Association for Improving the Condition of the Poor. This association wished to ascertain whether it is possible, when one or more in a family are afflicted with tuberculosis, to treat the patients in their own home, without danger to others; whether the results of treatment at home compare favorably with results obtained in sanatoriums, and whether, when tuberculosis and poverty are combined, it costs more to treat the family as a unit than if it is broken up.

The plan of operation has been to place in a sanitary tenement (having sunlight and fresh air in abundance) families selected because one or several members are tuberculous; to keep the family together, using whatever was earned by its members and supplementing this to permit of proper rest, sunlight and fresh air for those who were ill and a decent home for those who were well.

The Home Hospital, located at Seventy-Eighth Street and John Jay Park, occupies two entire sections of the East River Homes. Two open staircases lead to forty-eight apartments, each consisting of from two to four rooms, one or more being bedchambers with open air sleeping balconies. On the roof is a spacious solarium with hedges of privet and geraniums. A part of this solarium is reserved for the patients, another for a children's playground, another for a fresh air school.

The medical treatment has been that of any first rate sanatorium — rest in the open air and extra nourishment, tuberculin in a few cases. Of forty-five adult patients 46.6 per cent. have been cured, 35.5 per cent. have had their disease arrested, 17.7 per cent. have been improved — 99.8 per cent. improved in all. Among the children the improvement was particularly good. Not one unafflicted member of any family has shown signs of the disease.

Seventeen families have been rehabilitated physically and their earning capacity restored or even increased; and through the instruction they received they are no longer likely to be a menace to the community, but active centers in spreading a knowledge of hygiene.

Tables of comparative expense show that this method has cost less than any combination of sanatorium and institutional treatment, while it preserves the family unit and its earning capacity.

S. C. Kingsley[78] describes the open air school movement in Chicago. In the school year 1913-14 there were twenty open air schools in Chicago, with an attendance of over 600 children. The Board of Education furnished teachers and school equipment and the Municipal Tuberculosis Sanatorium of Chicago operated in nursing service and

food. The Elizabeth McCormick Memorial Fund employed special physicians, furnished clothing and gave other necessary service. The children selected were of the undernourished, tuberculous type and came mainly from the congested districts of the city. Seventy-five of the children were tuberculous; 20 per cent. were suffering from anemia and malnutrition; 5 per cent. from bronchitis, heart disease, etc. In addition 188 had diseased tonsils, 165 had adenoids, 466 decayed teeth, 365 defected eyesight.

In order that careful medical work might be done, the Elizabeth McCormick Memorial Fund has paid the physicians of the open air schools, and these physicians have spent an average of four hours a week in each of the nineteen schools. They find it takes a half hour to make a careful examination of each child; and such examinations are made on admission, on discharge and once or twice during the year. In addition, there is a daily supervision of each school. The nursing service takes about an hour and a half a day for each school. The nurse inspects the children daily, supervises the taking of temperatures, visits the homes, and gets the best possible help from the school for the child. Each teacher has only a small number of children in charge, so can know them well.

The children in these schools gain in weight, increase in alertness, in spirit and interest, and make intellectual progress.

C. Dinwiddie[79] describes the Bamford Hills camp for anemic and pretuberculous children, which is connected with the work of the Cincinnati Antituberculosis League. The camp is situated in a grove near the Miami River. The children are cared for by trained nurses, according to instructions given by the medical director. Their personal habits, diet and recreation are carefully supervised and they are taught the essentials of personal hygiene.

F. S. Minns[80] describes the method of dealing with tuberculosis in the public schools of Toronto. Medical inspection began there in 1910. At present, the total staff consists of a chief medical officer, twenty medical inspectors, one of whom is a special examiner for tuberculosis, one chief dental officer, thirteen dental surgeons, one superintendent of nurses and thirty-five nurses. All known cases of tuberculosis are recorded and every child suspected of having the disease, or of having been exposed to it, is examined by the special medical examiner. The medical inspectors and nurses assist the special examiner by referring to him all children exposed to, or suspected of having the disease, and by making a report of the home conditions. A record of every child examined for tuberculous infection is kept in the office of the chief medical officer.

Examinations made in the schools are arranged for by the school nurse, who obtains the parents' written consent to the examination.

The work of the department is carried on in conjunction with the work of the family physician, dentist, chest clinics at the hospital, the various missions, the social service organizations and the board of health.

Children examined are placed in one of three groups—those with positive, those with negative and those with doubtful evidence of tuberculosis. Those in the doubtful group are kept under observation and reexamined until placed in either the first or second group. The positive cases are subdivided into two groups, the open and the closed cases. Children who give a positive tuberculin reaction and show clinical evidence of tuberculosis are designated as closed cases. Any of these children who show defects, such as sore eyes, bad teeth, enlarged glands, etc., are referred to the family physician, or to proper authorities for treatment. Where tuberculin treatment is advised, it is given by the family physician or at the Hospital for Sick Children. A large number of the children with closed tuberculosis are sent to a preventorium for one to four months. During the summer, the Board of Education maintains two forest schools for children with closed tuberculosis.

Children with open tuberculosis are excluded from the schools and are visited by the Board of Health nurses. In some instances they are referred to the hospital.

A. Maggiora[81] outlines his ideas as to what the school should do in the combat against tuberculosis. He thinks it the duty of school authorities to conserve the health of its pupils, to build up the delicate ones, to diminish in them the disposition to disease and to propagate the idea that all members of society should contribute to the defense against tuberculosis.

The schools can do this by the following means: A competent physician should examine all children on admission and should make repeated examinations during the school year, in order to exclude any child with open tuberculosis. He should examine the entire personnel of the school for this disease. A separate department, preferably with open air class rooms, should be operated for delicate children. Convalescents from the acute infectious diseases and brothers and sisters of children suffering from the acute infectious diseases should be excluded from school. The locality of the school building, its construction and equipment should answer modern hygienic requirements. The cleanliness of the school should be minutely supervised. The curriculum should be arranged not to overtax the strength of the pupils and athletics should play an important part. A refectory should be conducted, especially in winter, and special nourishment should be provided for the delicate children.

In all schools there should be instituted propaganda against tuberculosis, and all teachers given instruction in hygiene.

Thiele-Chemnitz[82] deplores the law which excludes tuberculous children from school without making other provision for their education. He quotes Kirchner, who says that to forbid a child to attend school is to close the future to him. Tuberculous children should be placed where they cannot harm others, yet they should not be deprived of means of development.

The ideal is a sanatorium and school combined, in which the curriculum is arranged not to overtax the child.

A. Wallgren[84] emphasizes the importance of exposure to tuberculosis in the family during childhood. Among 100 tuberculous persons, he found in 51 per cent. a history of exposure to tuberculosis in the family before the age of 15; among 100 nontuberculous persons, in only 13 per cent. A history of exposure before the child was 5 years old was ascertained in 15 per cent. of the tuberculous and only 1 per cent. of the healthy children. Among the 100 tuberculous persons, one or both parents in 27 per cent. had been tuberculous; a brother or sister in 6 per cent., and other relatives in 11 per cent. Among the 100 healthy persons, only 2 per cent. had a history of tuberculous parents; 2 per cent. had a tuberculous brother or sister, and 2 per cent. other tuberculous relatives.

Thiele[85] compared the height and weight of 1,000 normal school children with that of 300 tuberculous children. He found that on entrance to school, tuberculous boys and girls weighed less than the normal children, and that the boys were also somewhat smaller, and manifested a marked retardation in weight and height throughout the entire school time. In the girls the failure to gain sufficiently in weight and height lasted until the middle of their school time. Then there was a rapid gain in height without a corresponding gain in weight. This gave rise to the "phthisical habitus" which, as is well known, appears later in boys than in girls.

Tuberculosis then has a detrimental effect on the development of the child, a fact to which too little attention has been paid.

W. L. Moss[86] has attempted to immunize calves against tuberculosis by feeding them the milk of cows vaccinated against the disease. He says it has been proved that an efficient immunity is established against a number of infectious and toxic agents; that, in the case of most of these, immune bodies can be demonstrated in the blood; that these immune bodies may appear in the milk also; that from the milk they may be absorbed from the alimentary canal of the infant and appear in the blood, where they exert a definite protective influence. He says, moreover, that active immunity against tuberculosis has been established in cattle, and he quotes v. Bering who has suggested the possibility of conferring passive immunity on infants by giving them the milk of vaccinated cows.

Moss performed two series of experiments. In each six calves of approximately the same age, weight and breed were selected. Three of these calves were from birth fed on fresh mixed milk of several vaccinated cows. The other three calves were fed with milk from unvaccinated cows. To vaccinate the cows, four intravenous injections of a suspension of living human tubercle bacilli were given at intervals of three weeks. After several months a virulent strain of bovine tubercle bacillus was used as a test inoculation. In the first series 1 c.c. of the suspension was inoculated into the anterior jugular vein. After the test all the calves rapidly developed tuberculosis and died during the fourth week. Evidently the dose was excessive and masked any evidence of immunity which might have been present in the calves fed on milk of vaccinated cows.

In the second series, the same dose of virulent tubercle bacilli was given subcutaneously. The three calves receiving milk from the immunized cows throve better than the other three, and at the time of necropsy weighed 25 per cent. more. A month to six weeks after the injection both groups developed lesions at the site of inoculation. In the calves receiving the milk of immunized cows the lesions were small and difficult to palpate, while in the other group they were very evident. The calves were killed and at necropsy tuberculosis lesions were found to be much more marked in the nonimmunized animals.

The blood of the calves of the two groups was tested for tuberculo-antibodies but no difference could be determined by means of the agglutinin, precipitin or complement binding tests.

The author thinks the conclusion justified by the results of the second series, that a relative degree of immunity against tuberculosis may be conferred on cows by feeding them the milk of vaccinated cows. Further experiments are necessary to determine whether human beings are similarly influenced.

TREATMENT

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93. Chadwick, H. D.: Treatment of Tuberculous Cervical Adenitis, Boston Med. and Surg. Jour., 1915, clxxii, 5.

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95. Meyer, L. B.: Conservative Treatment of Tuberculous Glands of the Neck, Med. Rec., New York, 1915, lxxxviii, 65.

I. O. Woodruff,[89] discussing the care of children suffering from tuberculosis in the early stages, finds the necessary preliminary step to

be accurate diagnosis. In this, familiarity with tuberculosis as manifested in the adult is of little value. But when it comes to treatment, important, as with the adult, are rest, fresh air and superalimentation. Rest especially should be emphasized. The orthopedic surgeons have long appreciated the necessity of absolute rest of the affected part in tuberculous bone and joint disease, so long as the process is active, and it is the writer's experience that rest is quite as essential in pulmonary disease. While the temperature in children does not seem to be affected by the toxemia or by exercise, the pulse is particularly susceptible, and it is well to base the amount of exercise in which the child may indulge on its condition. As the child improves more and more exercise can be introduced. In the sanatoriums and camps, a children's garden can be used admirably for this purpose. Exercise must be graduated, the pulse and temperature noted before exercise and one half hour after it, and the work graded according to its effects. Mentally satisfying the child with quiet occupation is important and simplifies the problem of restricted exercise. Schooling, always in the fresh air, may be permitted. Rest in a reclining posture should be insisted on for one hour after the midday meal. When the disease is quiescent or in an apparently arrested stage, postural and breathing exercises exert a marked benefit.

The value of fresh air is well established and the nearer to twenty-four hours a day it can be had, the better. It is fresh air rather than the air of any particular locality which is valuable, though an equable climate with fairly cool temperature, no dust or high winds is most satisfactory. A particular bad combination is heat and humidity.

As to diet, foods of high caloric value are indicated, until the weight is normal or slightly above. The type of diet is one high in protein and fat. Digestive disturbances are less likely to occur, if superalimentation is brought about slowly.

Of the adjuvants to rest, fresh air, and feeding, Woodruff mentions tuberculin. He uses it only for those patients who do not improve under ordinary remedial measures. He employs old tuberculin in very small doses. In cervical gland tuberculosis the writer is distinctly enthusiastic over the use of tuberculin. Surgical measures are indicated only where caseation exists or where the glands are present in large numbers.

A tuberculous child may be permitted to resume normal life when the pulmonary signs have entirely disappeared or have remained stationary or inactive for a period of months; when nutrition is normal; when the pulse is of normal rate and range and of good stability.

Woodruff lays especial stress on the after care of the child who has had an active tuberculosis, and on the prophylaxis of the disease. The fresh air class, he says, is the best avenue by which a child may

return to normal life. A child cured of the disease should sleep constantly with the window open or, if practical, out of doors. Each child should be discharged on probation, and at first should report once a month for examination. Later the period can be lengthened.

As a phase of after care, attention should be paid to the condition of general nutrition and chest development. Periods of rapid growth should be carefully watched. Whooping cough and measles should be guarded against.

In regard to prophylatic measures, Woodruff quotes figures showing that bovine tuberculosis is a menace important enough to be seriously considered. Raw milk can be relied on only when obtained from certified herds under proper supervision. Where standards of supervision do not prevail the public must be impressed with the necessity of pasteurizing the milk, or, better still, if its use is temporary, of boiling it, when intended for children.

The prevention of infection from human sources presents many problems. Too intimate contact with the infected member of the family must be prevented. Young children who creep or play on the floor must be excluded from the patient's rooms; carpets must be abolished; dustless methods of cleaning must be employed; washable rugs or rugs covered with muslin used. In municipal work, preventive measures include the iteration and reiteration of the necessary of care; the provision of a separate sleeping room for the adult patient, and the removal of the patient, if necessary, from the family; the sending of children at intervals to preventoriums.

If infection has taken place, the problem is to prevent its extension and development. This includes improving the state of nutrition, correcting defective posture and developing the chest by appropriate exercise; correcting of various physical defects, especially dental caries, hypertrophied tonsils and adenoids. All exposed children should have a cutaneous test. Children who do not react should be tested every four or six months. Children under 3 or 4 years who react to tuberculin are in need of medical care and are especially suitable for preventorium treatment.

H. Koch[99] has given tuberculin rather extensively in the treatment of tuberculosis in children and believes success depends on the technic of its administration. He reports his experience in the treatment of forty-five cases. He used only two preparations, Koch's old tuberculin and the albumin-free tuberculin. The initial dose was $\frac{1}{1000}$ mg. of old tuberculin and $\frac{10}{1000}$ mg. of the albumin-free tuberculin. This dose was increased in geometric progression, until doses of 1,000 mg. of old tuberculin and 10,000 mg. of albumin-free tuberculin were reached. The number of doses between the initial and final doses were seven (rapid increase) thirteen (medium) or twenty-five (slow). On an

average, however, the end-dose was reached by eighteen injections or in nine weeks, as two injections were given each week. The cause of this was a necessity of repetition of the same does or the return to a smaller dose, on the one hand, and the skipping to a larger dose, on the other. In the writer's cases delays due to repetition of doses were comparatively rare. The end-dose was repeated in a number of instances.

Both preparations of tuberculin were given well diluted with physiologic salt solution (10 to 20 cm.). This large amount of fluid seems to aid absorption and allows of a general distribution of tuberculin throughout the body and a general formation of antibodies.

The injections were given between the shoulder blades and never twice in the same place. In this way reactions were avoided. Reactions from injections of tuberculin may be general or local. Of the general reactions the most important was fever. In nineteen of the cases there was undoubtedly a febrile reaction. It occured eleven times with normal increase of dose, five times after repetition of the dose and five times with a rapid increase of the dose.

Inflammatory manifestations at the site of injection occurred in seventeen cases: in eight cases once, in six cases twice, in two cases three times and in one after every injection. This last case was not suitable for tuberculin treatment.

In seven cases the tuberculin cure was interrupted. In three of these there was a wide-spread infiltration of the lung with cavity formation; in two cases, one of scrofula and one of tuberculosis of the skin, the local reaction was too severe to continue and in two cases of multiple tuberculosis of the glands the course was not influenced.

Good results were obtained in the other thirty-eight children. Their general condition improved, the fever either disappeared or was less severe and the tuberculous process was favorably influenced. In two cases of pulmonary tuberculosis there was retrogression of inflammatory manifestations, the tubercle bacilli disappeared from the sputum and there was a partial cessation of cough.

Only a few forms of tuberculosis in children are not suitable for specific treatment. To these belong severe phthisical processes, amyloid degeneration of the parenchymatous organs, cases with marked reactions to tuberculin, cases of miliary tuberculosis and of tuberculous meningitis.

The good results which Koch has obtained with tuberculin makes him recommend its use in tuberculosis of childhood.

In spite of the fact that experimentally J. Bacigalupo[92] could not be convinced of the penetrability of various substances into the serous cavities under normal and inflammatory conditions, he determined to

try the effect of intradural injections of tuberculin in cases of tuberculous meningitis.

The first case was one of advanced tuberculous meningitis, complicated by miliary tuberculosis. Twenty-four hours after the injection there was some amelioration of the brain symptoms, but the child died three days later. Bacigalupo gave the same treatment in two uncomplicated cases, in both of which tubercle bacilli were found in the spinal fluid. In both the results were very satisfactory, and there was complete recovery after two or three injections in the course of twenty days. Worthy of note is the fact that there was no reaction to the tuberculin in any of the three cases.

As regards dosage, in all three cases the first dose was 1 mg. followed by a somewhat larger dose in twenty-four hours, if there was no improvement in symptoms.

As the author finds only twenty-two recoveries from tuberculous meningitis reported in the literature, he thinks it is worth while to try further intradural injections of tuberculin.

H. D. Chadwick[93] describes a method of treating children admitted to the Westfield Tuberculosis Sanitarium. Most of these have tuberculous cervical glands and also enlarged bronchial glands, as evidenced by impaired resonance between the scapulae. Chadwick puts these patients on bacillen emulsion, if they do not have more than a degree of fever or show signs of active pulmonary disease. The initial dose is one-millionth of a milligram and is increased in the course of six months until the maximum 10 mg. is reached. This maximum could be reached in a shorter time, but a small dose given over a longer period is more effective and does not cause reactions.

The results of treatment in these children is very satisfactory. The cervical glands decrease in size and the area of dulness over the hilus becomes smaller. Suppuration never occurred, when it had not existed before treatment. This experience leads the author to the conclusion that surgical interference is necessary only to remove such glands as have become caseous or fibroid.

G. L. Richards[94] discusses the relation of tonsils and adenoids to tuberculous cervical adenitis. There are two principal retropharyngeal glands, one on either side of the median raphé. These glands receive lymph from the mucous membrane of the nasal fossae and adjacent cavities and drain into the upper glands of the internal jugular chain. The general lymphatic drainage of the pharynx also ends in the retropharyngeal glands and the internal jugular chain. The glands of the internal jugular chain receive also the lymph from the internal group of the sternomastoid glands.

The lymphatics from the tonsil drain into the lymph glands of the tongue, thence into the two lateral trunks, which pass down the

lateral wall of the pharynx and terminate in the large glands of the internal jugular chain. A not inconsiderable number of lymph vessels go directly from the posterior pharyngeal wall to the deep glands of the neck and internal jugular region.

The adenoid area drains into the posterior wall of the pharynx. The lymphatics of the larynx drain into the glands of the internal jugular chain.

Richards thinks that after the removal of the faucial tonsils in children suffering from enlarged cervical glands, these glands disappear and are no longer palpable. He recommends in all cases of cervical adenitis that the tonsil be removed as the first procedure, but he does not assert that the removal of the tonsils removes all danger of infection of the cervical lymph glands, since the anatomic studies referred to show that the area of the middle ear, Eustachian tube, superior, posterior and lateral pharyngeal wall, the tonsils, base of the tongue, and posterior nares, all drain into the cervical lymph glands.

L. B. Meyer[95] believes in the conservative treatment of tuberculous cervical glands and he particularly emphasizes the importance of correcting in the mouth and nasopharynx all pathologic conditions which might act as a source of infection. Diseased tonsils and adenoids should be removed and the proper treatment for decayed teeth be instituted. This accomplished and the various hygienic and tonic measures recommended for tuberculosis carried out, a large majority of patients with tuberculous glands will probably never need surgical treatment. If, however, the glands do not disappear under this régime, they often will if treatment is directly applied to them. Of these Meyer mentions injection of 5 per cent. iodoform emulsion in oil, or 2 to 4 per cent. formaldehyd solution in glycerin, the use of Biers hyperemia, of tuberculin, and Roentgen ray.

If the patient is not seen until a glandular abscess is formed or a sinus exists, treatment must be directed to prevent the formation of disfiguring scars. Often appropriate treatment applied to throat and mouth is followed by retrogression of the glands and healing of the sinus. If not, a little assistance with a curet or stimulation with tincture of iodin, or the use of Beck's paste is all that is needed. If an abscess exists, the pus must be evacuated either by aspiration or with a narrow knife. It is usually better to wait, in this condition, for complete caseation of the gland.

Meyer cautions against extensive dissection in these cases.

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